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FRONTAL LOBECTOMY IN THE TREATMENT OF BRAIN TUMORS*

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FRONTAL LOBECTOMY in the radical treatment of brain tumors is of twofold interest. To the neurophysiologist it furnishes an approach to the study of frontal lobe function, supplementing clinical observation and experimental investigation. To the surgeon it is of primary interest from the standpoint of the surgical removal of brain tumors. It is from the latter point of view that the present report is made. The surgical aspects, however, here as elsewhere, are inseparably bound up with the consideration of function—its disturbance by the lesion and the possibility of its restoration by adequate therapy.

Starr,[16] as early as 1884, reviewing 23 cases of frontal lobe lesions, found lack of self-control to be the most common feature, with inability to fix the attention, to follow a continuous train of thought, or carry on intellectual processes. To this loss of self-control he attributed the associated changes in behavior, since the mind exercises a constant inhibitory influence upon all action, physical and mental, from the restraint of the lower reflexes to the higher control of emotional impulses and their manifestation in speech and expression. The exercise of control implies a recognition of the import of an act in connection with other acts. In a word, it involves judgment and reason. By inhibiting all but one set of impulses, it makes it possible to fix the attention upon a subject and to hold it there. Starr regarded it as probable that the processes involved in judgment and reason have for their physical basis the frontal lobes. Destructive lesions of these lobes, therefore, would manifest themselves by errors of judgment and reason of a striking character.

Starr's observations were borne out by those of Mohr[13] on a case of pituitary tumor causing compression of the frontal lobes, manifested by childishness and urinary and fecal incontinence. Another early example was the famous "crowbar case" of Harlow[9] (1868), in which the patient suffered severe damage to the dominant frontal lobe. From a hard-working foreman,

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commanding the respect of both his employer and workmen, he became fitful, impatient, capricious and vacillating, and a child in intellectual capacity.

Since these early reports a vast literature on frontal lobe tumors has accumulated. The more striking features, as recorded by others and as observed in our cases, are disturbances in memory and orientation, changes in personality, inability to synthesize thought processes into more complex reasoning, and *"witzelsucht."* Less common are the so-called frontal lobe stupor and vesical incontinence. Holmes,[10] in 1931, described three types of personality change: A type characterized by apathy and indifference; another by depression, automaticity, and incontinence; and a third by restlessness, exuberance, euphoria and *"witzelsucht."* All three types were observed in our series. The individual personality probably determines in some measure the degree at which these various types are unfolded with the dissolution accompanying the disease process.

The frontal lobe, defined as the area anterior to the central sulcus, includes four subdivisions: the motor area, Area 4 of Brodman;[2] the premotor area, Area 6 of Campbell;[3] the speech area; and the frontal area, the region anterior to Brodman and Campbell's Area 6. Area 6 has been further subdivided by Vogt[17] into Area 6 $a\alpha$ and Area $a\beta$, the former lying, in man, within the precentral convolution.

The term lobectomy is, we believe, misleading. It has been used, however, by Dandy,[4] Spurling,[15] Penfield and Evans,[14] German and Fox[8] and others, to designate removal of the frontal area exclusive of the speech, premotor, and motor areas, and is often spoken of as the prefrontal area. The line of incision in our series has, as a rule, included Areas 8, 9, 10, 11, 32, 34, 46, and 47, and occasionally 6 $a\beta$ but never 6 $a\alpha$ or 4.

McCaw,[12] in 1919, reported a case of resection of the right temporosphenoidal lobe, without, however, a detailed study of the after-effects. Dandy, in 1933, stated that the entire right or left frontal lobe could be removed "without any observable mental or other after-effect."

Critical studies of the after-effects of unilateral frontal lobectomy have been published by Penfield and Evans, German and Fox,[6, 8] and Spurling. Penfield and Evans, in 1932, reported four cases. In one of these the operation was undertaken for removal of a calcified oligodendroglioma, in the other three for posttraumatic convulsive seizures. German and Fox,[8] in 1932, resorted to lobectomy in two cases of spongioblastoma, and Spurling, in 1934, removed the right frontal area for a subfrontal meningioma. Thus, of the seven lobectomies in which a critical analysis has been made, three were performed for tumors of the glioma series, three for cerebral scars, and one for a subfrontal tumor.

The case material forming the basis of this discussion consists of 11 gliomata of the frontal lobe. Cases in which the operation was performed for traumatic epilepsy and a small series of meningiomata will not be considered here.

This last group presents certain practical problems which have not yet been satisfactorily solved, at least in our hands. Fatalities have occurred in this

latter group which we believe can be avoided in the future as we have further opportunity to master the problems involved. The operation for these tumors consists of two major procedures: The removal of the frontal lobe, which in itself is an operation of considerable magnitude; and, following this, the difficult attack upon the large meningioma lying in close proximity to the great vessels at the base and in part surrounding the vessels forming the circle of Willis.

Of the 11 gliomata, three were in the glioblastoma group, and nine in the astrocytoma series. All of the patients survived the operation and, with the exception of the three who had extensive glioblastomata, all are alive and carrying on useful lives. In six of the nine patients with astrocytoma, we were able to excise the tumor completely and, we hope, effect a cure. In the remaining three the tumor extended beyond the line of excision, either dorsally into the motor area or across the midline into the corpus callosum. In the following discussion we shall consider chiefly those cases in which the lesion was completely excised, though the three patients in whom tumors still remain form an interesting series. They have shown some improvement and have made fair social adjustments.

It has seemed curious that though unilateral frontal lobe tumors, with but little evidence of generalized increased intracranial pressure, give rise to symptoms permitting their diagnosis, yet the most thorough and painstaking clinical studies are essential to detect absence of the frontal lobe after its removal. A far more searching examination is necessary to discover that frontal lobectomy has been performed than to determine the presence of a frontal lobe tumor.

The frontal lobes are primarily association centers receiving impulses from all other parts of the brain. They are intimately connected, so that the proper function of each is dependent upon impulses of a certain order and frequency reaching it through the association paths between the two.

According to Hughlings Jackson[11] the muscles of each extremity have bilateral representation in the dominant hemisphere and also in the hemisphere of the opposite side. This he held to be true not only for muscles acting separately but more especially for muscles which function synchronously, as the facial muscles, those of the diaphragm, and others. Jackson states that: "My supposition is that the limbs of the two sides are very unequally represented in each half of the brain while the bilaterally acting muscles are very nearly equally represented in each half." If this be true for muscles, how much more likely is it that syntheses which constitute personality, thought, memory, *etc.*, must be the combined product of both frontal lobes, and not of one or the other alone, regardless of the handedness of the individual. There would seem to be little doubt that personality and the association and synthesis of mental processes are bilaterally represented and that their expression is the simultaneous product of both frontal lobes.

The presence of a tumor in either lobe distorts the frequency and type of wave discharge, interfering not only with function on the diseased side but

also on the opposite side. Frontal lobe symptomatology is thus expressive of disturbance of both lobes and not of one alone. The smooth performance of either lobe is dependent upon the smooth performance of the two together. If distorted impulses from one lobe reach the opposite side, disturbance of function becomes evident. If, however, the disturbing lobe is removed, whether this be dominant or nondominant, as has been done in the cases recorded here, the remaining lobe is able to carry on so well that the loss is hardly to be detected.

The "static," so to speak, produced in one frontal lobe interferes with the function of the opposite lobe as well as with its own. Normally, both frontal lobes function together harmoniously, or one may compare the function of the frontal lobes to a well synchronized and harmoniously playing orchestra. If the first violins play out of tune, or play their own tune, the music produced by the orchestra can hardly be called music. If, however, the first violins are removed the orchestra may well go on playing in harmony as a united symphony. By removal of the diseased lobe the irritative lesion is eliminated and the patient is left with an intact frontal lobe which is capable of carrying on now that it is no longer disturbed by irregular and purposeless emanations from the opposite side. This applies not only to removal of the nondominant, but also the dominant frontal area. In either instance the remaining lobe is able to carry on so well that the absence of the other is difficult to detect. This concept of frontal lobe function, considered in its broadest sense, is borne out by the cases recorded here.

A critical search for frontal lobe signs following lobectomy has yielded little of importance. The grasping and groping reflex described by Adie and Critchley[1] was not present in a single instance. It will be recalled that in their review of the cases in the literature, these authors concluded that the lesion responsible for this reflex was situated in the posterior portions of the superior and middle frontal convolutions. Their figures indicate approximately the Area 6 $\alpha\beta$. When the grasping reflex is present, it is taken to indicate a lesion in this region, but certainly large tumors of the frontal lobe may exist in which the reflex is not present. In those instances, furthermore, in our series in which this area was traversed by the line of excision, the reflex did not occur. This was also the experience of Foërster, who reported excisions of this area without detectable signs. His explanation is in accord with Hughlings Jackson's suggestion that if the movements produced by one area are represented in another area, in approximately the same combination and order, they need not be lost with removal of the area in which they are produced.

Foërster[5] found that the movements produced by Area 6 $\alpha\beta$ were similar to those elicited by Area 5. The inference is that at least part of the function of Area 6 $\alpha\beta$ is taken over by Area 5. This being the case, one would expect to find the grasping and groping reflex or some modification of it, in lesions of the latter area.

It is also noteworthy that in some cases reviewed by Adie and Critchley

the lesions involved the basal ganglia. The area outlined by those writers has been shown by Fulton and his coworkers[7] to be essentially the region which they have demonstrated as the cortical representation of the extrapyramidal motor system.

The study of the patients in this series has consisted of a complete neurologic examination and a social investigation, with observations on admission to the hospital and a follow-up by social workers who visited the patients at regular intervals. The early development, scholastic background, social and family life have been gone into thoroughly and information from other sources has been obtained. Psychiatric interviews were conducted by Doctor Teitelbaum, whenever possible, prior to operation and in every case following operation. Complete psychometric tests were made by the Psychology Department, Miss Tallman doing all the testing. These included vocabulary tests, performance tests, and personality tests, such as the Rohrschach and Jung association tests.

The conclusion reached is that the patients in whom the lesion was completely excised, whether from the dominant or nondominant hemisphere, were intact, both as regards the past and the present life. They appeared genuine in their responses and on nonpsychiatric investigation it would be exceedingly difficult to tell that there had been anything wrong with them. In several instances, indeed, they functioned on a better level than before operation. They showed little impairment of general intelligence, they planned and executed household duties competently, were able to sew, market, cook, plan for guests, *etc.* There was no slowing-up of the common everyday performances. In some instances a lack of distractability was notable. There was no hesitation in putting into effect a decision, but the decision once made was maintained until its execution was completed.

There was a complete return of the former emotional tone. The sexual life of five or six patient returned to its former status. In one, however, frigidity developed and she came to look upon the sexual act solely as a duty.

Some of the patients showed distinct evidence of a new ability to learn; they acquired new interests, became socially more adaptable, and calmer emotionally. None showed any loss of inhibition, and in not one was there any disturbance of spatial orientation.

However, in the group in which the tumor extended beyond the line of incision, the results were quite different. These patients were shallow emotionally, listless, indifferent, and lacking in initiative. One, though she was able to care for herself, sat about the house day after day, refusing to go out, and evincing no interest in outside life. Another, while markedly improved in intellectual functions, showed a failure in social adjustment and a lack of inhibition, wetting herself, for example, and attributing this to the poisoning of her food by others. Three of the patients in this group, however, were able to care for their homes and children, and to adjust themselves mentally so long as no complex situation arose. They were, for example, able

to talk intelligently with one or two people, but were unable to carry on a conversation with a number of people or in a crowd.

This series of cases thus offers confirmatory evidence of the theory suggested earlier in this communication. Where the lesion was completely removed, the intact lobe freed from the distorted impulses due to disease of the opposite side proved able to carry on for all practical purposes as efficiently as before. When, on the other hand, only partial removal was effected, though some improvement took place an irritant still remained, interfering with the smooth synchronous functioning of the two lobes.

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DISCUSSION.—DR. JOHN E. SCARFF (New York) said that as a result of the studies by Doctor Stookey, Doctor Teitelbaum and himself, it would appear possible to make certain generalizations regarding function—and the

functioning—of the frontal lobes: In the first place, it would appear that either one of the frontal lobes, singly, is capable of carrying on in a satisfactory manner the work normally done by the two frontal lobes working together, provided that the remaining frontal lobe does not have its function interfered with by any disease process. This interference with function might come from extensions of the tumor remaining outside the lines of excision of the lobe, either on the side of the lobectomy or by invasion of the opposite side by way of the corpus callosum.

The second generalization is that it was not possible to find any dominance existing in the frontal lobes. It is well known that dominance exists in the temporal and parietal lobes, and it has been generally assumed that this existed in the frontal lobes. Observations made in the present study did not confirm this general concept. If there was a clean removal of the left frontal lobe the patient did as well afterward as did the patient from whom the right frontal lobe had been removed. There were no exceptions to this rule in this series.

In the third place, a better idea of the mechanism by which the so-called "frontal lobe syndrome" is produced has been acquired. This syndrome is characterized by defects in intelligence and alterations in personality. It would seem that this "frontal lobe syndrome" is due not so much to *suppression* of frontal lobe function as to *distortion* of frontal lobe function, by some pathologic process. Doctor Stookey used the simile of an orchestra to illustrate this point, and indicated how one section of violin, playing out of key, would result in discord from the efforts of the entire orchestra; whereas, if the off-key violins were removed, the remaining portions of the orchestra would be able to produce pleasing music.

Doctor Scarff had hoped Doctor Stookey would find time to mention one patient who illustrated this last point very well, namely, that it is not the absence or suppression of function but the distortion of function that is responsible for the "frontal lobe syndrome." A woman, in her thirties, had for many years supported herself as a commercial artist, and was very successful. Then she became ill. When seen on the ward, she was quite sick and was confined to bed. She appeared alert and followed the examiner with her eyes and smiled. She had no true motor weakness but was completely helpless. Objects would be placed in her hands and she would not know what to do with them. A tray would be placed in front of her with luncheon on it, and eating utensils were there, but she could not figure out how to get the food to her mouth. Although she had power in her extremities she could not stand alone. In short, she was completely apraxic. Although there was no sign of generalized intracranial pressure, study showed that she had a left frontal lobe tumor. Following its removal she became practically a normal person within a very few weeks, and was soon able to draw quite quickly and accurately, and as well as before. It would seem as though "static" given off by the diseased left frontal lobe had caused interference with the smooth functioning of the normal right frontal lobe; and that after the removal of this "static" the right lobe carried the load quite satisfactorily.

The cases that have been shown were all gliomata. When the tumor was removed, as a matter of course, the frontal lobe came out with it. There is, however, another group of tumors which are more benign and are encapsulated, located not within but underneath the frontal lobe. In the past, neurosurgeons have been impeded in their attack upon these tumors by inability to retract the frontal lobe adequately enough to give them a good exposure. The result was that, often, for this reason only, these tumors could not be completely removed. Adequate exposure could have been obtained if the frontal lobe were removed. Surgeons have hesitated, in the past, to do this because they

felt by so doing they would leave the patient with an intellectual deficit. The present studies suggest that these subfrontal lobe tumors may be uncapped with safety, and the authors are already working on a group of cases having this type of frontal tumor in which the first step in the operative attack is clear exposure of the tumor by means of frontal lobectomy.

DISCUSSION.—DR. JOHN TEITELBAUM (New York)

A. THE APPENDED FINDINGS MAY BE REPORTED ON GROUP A, *i.e.*, THOSE IN WHOM THE TUMOR MASS WAS REMOVED COMPLETELY

(1) Complete *psychometric* and *psychiatric* studies showed little disturbance of function in this group. The nonpsychiatric observer would find it exceedingly difficult to say that there was *anything wrong* with these patients.

(2) *All patients were completely in contact with the past as well as the present life.* They are all able to function mentally, the same as they were before, and in the majority of cases on a much better level than before the operation. In conversation they make a good transference, discuss their problems frankly and openly. The study revealed that they are generous, kind-hearted, and interested in the welfare of their friends and families. Several of the patients have kept up an intelligent correspondence with some of the patients they met on the ward. They have retained their former abilities at cards, games, *etc.*

(3) The patients showed *no impairment of general intelligence.* They plan or execute business duties. One of them, three weeks after his return from the hospital, resumed his former employment, acting as inspector and taking care of his own account, as well as the accounts of eight men who work under him. All information gathered shows that his efficiency is as good as it ever was and that his mental functions are as keen as ever. He has to know by heart over 2,000 customers and keep their accounts; and if any of the eight drivers gets sick he is able to take his place as driver and make all deliveries correctly.

(4) The reactions presented by these patients are undoubtedly *colored by earlier drives*, and *personality* structure, as are all individuals. It would seem that their old patterns of reactions are either left the same or are modified in direction of more socially acceptable behavior.

(5) These patients have become *socially more adaptable* and *emotionally more calm.* The emotional adjustment was adequate and there was a complete return to their former emotional level. There was an improvement in their temper; they were less irritable, and more tolerant.

(6) All patients showed an *adequate response to humor.* The memory and insight into the feelings of others was unimpaired. In every case the humor was adequate and in good taste. One patient, who was always of a very serious nature and rarely showed much response to humorous conversation, now mentions jokes in her social groups, responds happily to comedy pictures (movies), and in every way has developed an adequate sense of humor.

(7) *The following changes which can be classified as being abnormal,* were noted. In two of the patients the examiner felt that there was a *lack of justifiable anxiety.* They were not concerned or worried about their future. They seemed to be perfectly satisfied to go on as they were. The patients of this group presented a *lack of distractibility* which was quite marked post-operatively, but which tended to diminish as time went on. When speaking or doing anything they could not very easily be distracted or turned to something new.

SUMMARY OF THE FINDINGS ON GROUP B, *i.e.*, THOSE PATIENTS IN WHOM THE LESION COULD NOT BE REMOVED COMPLETELY

The mental symptoms found in this group after observation, for 27 months, are still very numerous. None of them indicate any fundamental change in any mental processes, but only a disturbance of its completeness. The difference observed in these patients is a defect in the amount of this building process, thus limiting the degree of possible complexity of thought. The many symptoms to be enumerated flow from this deficiency, and as a result the personality becomes greatly altered.

On every-day tests these patients are able to function very well; some of them plan their meals, market, cook, bake, pay the bills, show interest in managing their home, and in taking care of their family. Some in this group show an improvement in character over their previous adjustments. A few, however, remain dull, lack initiative, efficiency, and planning. There is an emotional inadequacy. The feeling of the examiner is that they could not be pushed too hard in any task requiring more elaborate functioning. In other words, they are able to work on a simple level which is very likely slightly below the level under which they functioned prior to the development of the illness.

SUBDURAL HEMATOMA*

A STUDY OF ONE HUNDRED FORTY-THREE CASES ENCOUNTERED DURING A FIVE-YEAR PERIOD

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DURING the past 15 years a number of articles have appeared in the literature concerning cerebral subdural hematoma. Among these may be mentioned those by Putnam and Cushing[1] Gardner,[2] Peet and Kahn,[3] Jelsma,[4] Dyke,[5] Kennedy and Wortis,[6] Munro[7, 8, 9] Leary,[10] and Kunkel and Dandy[11] as representing noteworthy contributions relative to the pathogenesis, the diagnosis and the surgical treatment of this lesion. The publications of Donald Munro in particular have greatly influenced the trend of thought concerning the physiochemical changes that occur within and about a blood clot in the cerebral subdural space. Furthermore, he has repeatedly expressed the opinion that the chronic form of subdural hematoma is nothing more than a later variant of the acute phase, a concept with which we are in complete agreement. On the other hand, there is ample evidence to substantiate the opinion that subdural hematoma in the chronic form manifests different clinical findings from those evinced in the acute stage. In a series of cases here analyzed no attempt will be made to separate the acute from the chronic lesions. The period of time elapsing between trauma and verification of the clinical diagnosis in each instance has been tabulated, thus permitting the reader to arrive at his own classification.

From July 1, 1934, to July 1, 1939, 10,223 patients were admitted to the Neurosurgical Service of the Kings County Hospital with evidence of trauma to the intracranial structures. During the same period 42 patients with similar lesions were admitted to the Brooklyn Hospital. In the Kings County Hospital series there were 127 instances of subdural hematoma and in the Brooklyn Hospital group, 13 examples of this lesion. In addition three cases of subdural hematoma treated at the Long Island College Hospital have been included.

It is the object of the present study to consider the important observations made in these 143 cases of head injury which had as a complicating lesion, blood (fluid, clotted or organized) in the subdural space in sufficient

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amount to be of surgical significance (over 25 Gm.). All instances of surface brain laceration with a 1 to 2 mm. film of blood clot over and immediately about the laceration, although within the subdural space, have been excluded from the group under consideration. On the basis of the data derived chiefly from this study, the important aspects of the subdural hematoma will be discussed.

Etiology.—Trauma is the important agent in the production of subdural hematoma, although other causes have been encountered. For example, we have observed an extensive subdural hemorrhage from the region of a metastatic tumor implicating the pia-arachnoid, one from the vicinity of a cerebral abscess, two from ruptured cerebral aneurysms and one from an arteriovenous malformation on the surface of the brain. The classic pachymeningitis hemorrhagica interna of Virchow,[12] probably resulting from repeated small arterial hemorrhages, may also be included as an unusual type. In the group of cases here presented there were 133 in which the subdural hemorrhage was clearly the result of trauma, the site of the injury to the head varying considerably (Table I). As indicated, in approximately one-third of the

TABLE I

Trauma to the frontal area of the head	24
Trauma to the lateral area of the head	32
Trauma to the occipital area of the head	45
Trauma to multiple areas of the head	10
Trauma to unknown site	22
No trauma	2
Questionable trauma	8
	143

total number of cases there was evidence of a blow to the occipital region of the head. Most of the hematomata appeared to be venous in origin, although some resulted from arterial bleeding. There were examples in which no doubt could be entertained regarding the source of the blood clot, such as a rent in the dura with a tear of the meningeal artery caused by a fracture of the skull. A cerebral artery may be the source of the bleeding, seven cases having been observed in which there was a blood clot in the subdural space and in the adjacent cerebrum itself, both the result of a traumatically opened artery. Venous bleeding may occur from ruptured veins that cross from the cerebrum to the dura mater. A vein, almost constantly present, that may easily be torn as the result of trauma, is situated at the anterior aspect of the sylvian fissure and crosses the subdural space where it joins the sphenoparietal venous sinus (Fig. 1). Other veins observed to be the source of bleeding are those crossing or impinging upon the subdural space in the parasagittal region. These veins are more frequently traumatically opened in instances in which the cranial vault is grossly distorted, as occurs in the passage of the infant through the pelvic canal. Laceration and/or contusion of the surface of the brain implicating cortical blood vessels are much more commonly the origin of bleeding. In fact in our experience, fully 75 per cent of all subdural

hemorrhages can be traced to such a lesion. In some instances it is difficult, even at autopsy, to definitely identify the origin of the blood clot, particularly if several days have elapsed since the injury or when there are multiple areas of traumatized cerebral surface adjacent to the hematoma.

Pathogenesis.—In a discussion of intracranial lesions dependent upon trauma complicated by subdural hematomata, it seems permissible to incorporate certain general information acquired at autopsy on subjects other than the cases included in this series. Furthermore, the information amassed from

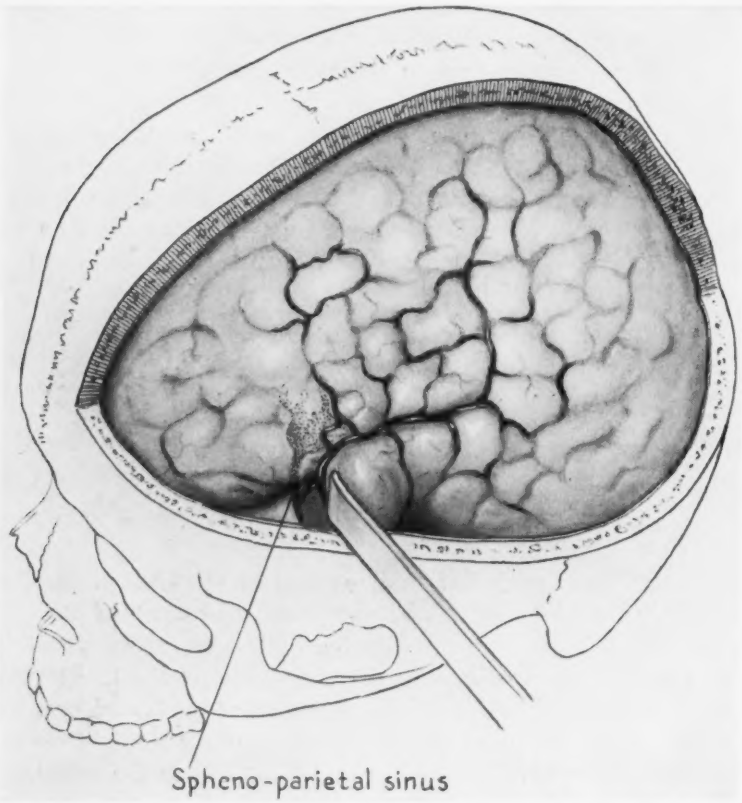

FIG. 1.—Drawing showing a vein not infrequently lacerated by trauma to the head. For illustrative purposes it was necessary to make the vein appear twice as long as observed in the average anatomic specimen.

the autopsy material is much more reliable than impressions gained from inspecting the intracranial contents through an operative opening. There is found great variation in the position, size and consistency of the subdural hemorrhages. In the series under review there were 71 hematomata in the left subdural space and 62 on the right side. In ten cases the blood clots were found bilaterally. Some were limited more or less to the temporal fossa, many were higher in position and thickest over the premotor area, while others covered almost the entire convexity of the cerebral hemisphere (Fig. 2). The lesion was encountered in the posterior fossa in two cases. The weight

of the clots varied from 25 to 350 Gm. The subdural collections were observed to be clots, fluid, organized masses or a mixture of these in various

FIG. 2.—A "classic" example of a fresh subdural hematoma.

TABLE II

Elapsed Time from Injury to Operation or Death	2 to 24 Hours	24 Hours to 4 Days	4 to 8 Days	8 to 13 Days	13 to 17 Days	17 to 26 Days	26 to 36 Days	36 Days to 3 Mos.	Total
Fluid blood	7	5	5	6	9	4	5	2	43
Clotted blood	17	8	10	2	1	1	2		41
Clots mixed with fluid	8	8	8	3	3	5	3		38
Clot organized					1			1	2
Hydroma		1				1			2
Insufficiently described									17

proportions (Table II). It may be noted from Table II that sufficient description was not recorded in the case histories to definitely classify the types of subdural collection in 17 instances. The time intervals used are the same as those employed by Munro.[9] The object of this selection was to compare the data of our series with those reported by him. The findings in our series indicate that large solid blood clots have seldom been encountered later than eight days after their establishment in the subdural space. In the unusual instances in which the hematomata remained solid after the first eight days, fibroblastic invasion of the clot was demonstrable. The findings further show that fluid blood, as well as the combination of clotted blood and fluid, was disclosed in a relatively large number of cases operated upon within eight days after injury. In general, it may be fairly stated that there was no gross pathologic evidence derived from the study of the types of subdural collections recorded in our series to refute the theoretic concept set forth by Gardner[2] and elaborated by Munro. Their theoretic explanation that a subdural hematoma may be augmented by osmosis in a certain number of such lesions seems entirely logical. There are other types of subdural collections in which the physical characteristics are so bizarre that it becomes difficult to interpret them in terms of this concept. These escape classification not only with reference to their formation and consistency but also as to the development of a surrounding membrane, subsequent changes within the clot and residual pathologic states. For example, in the series of cases that were operated upon on the fifth day following the injury, one finds such variations as collections of black glistening oil-like fluid, brownish-black coffee-ground-like material, solid liver-like clots, thick homogeneous tar-like blood and combinations of blood clot and thin, dark brown fluid. It, therefore, becomes obvious that there are factors governing the alterations that may occur in a subdural hematoma concerning which there seems to be no sound theoretic explanation. It might be suggested that the changes in consistency which take place in some hematomata are partly attributable to the variations in the rapidity and possibly the manner of formation of the membrane that eventually encases all fluid subdural collections. In some instances a definite membrane has been demonstrable as early as the fourth day, whereas in others in which the clots had the same physical appearance there was scanty or no membrane formation on the eighth to the tenth day following injury. In other examples in which no liquefication of the hematoma occurred, the cellular elements that comprised the membrane were relatively thin on the surface of the clot, and, depending on the age of the lesion, cells resembling fibroblasts were found to have invaded the hematoma to a variable extent. The membrane adjacent to the dura (outer membrane) has been observed in all cases to be thicker than that in apposition to the arachnoid (inner membrane); however, both membranes are seemingly formed at about the same time. The simultaneous development of an inner and outer membrane about a fluid collection tends to throw some doubt on the hypothesis that the dura is the sole origin of the cells forming the membrane. It seems unlikely that an outgrowth of cells

from the inner surface of the dura would proliferate with sufficient rapidity to cover the inner area of hematomata of the size encountered in this series, within four to five days. In addition, the presence of blood in the subdural space may not be a necessary factor in the production of a membrane about a subdural collection. A 60 cc. sterile subdural hydroma with well-established inner and outer membrane has been observed in association with multiple metastatic foci of infection within the brain (Fig. 3). Since the fluid of this particular hydroma was yellowish in color, it may be contended that

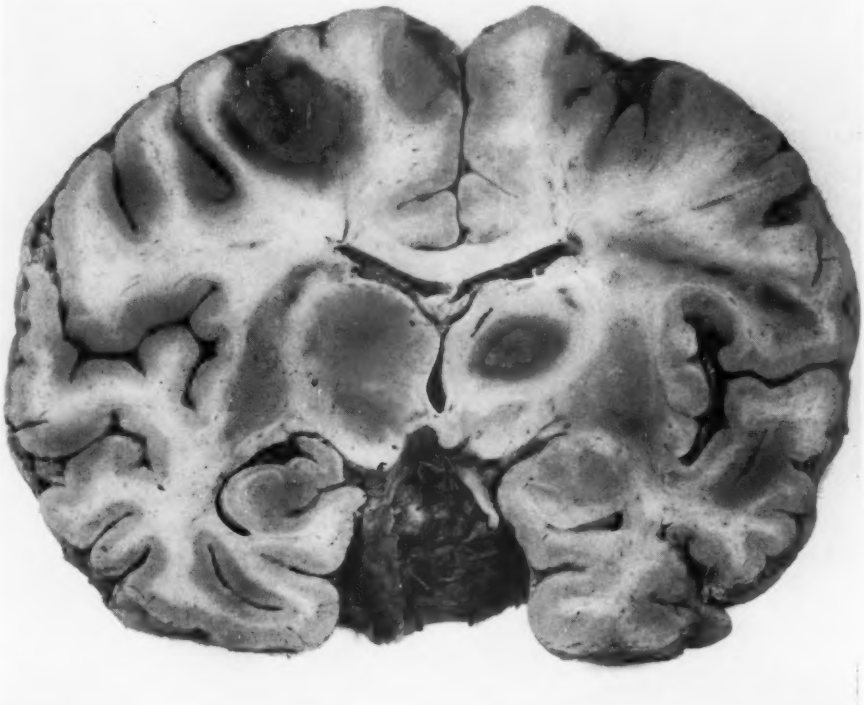

FIG. 3.—Section showing subcortical metastatic focus of infection associated with a subdural hydroma.

red blood cells or their derivatives were present and essential for the formation of the membrane. In general it may be said that most fluid subdural collections become enclosed by a cellular membrane, thicker on the outer (dural) and thinner on the inner (arachnoidal) side, that some solid clots become encapsulated and undergo central liquefication and that other solid hematomata are invaded by fibroblastic cells without evidence of dissolution.

The type of brain lesion sustained in association with subdural hematomata may also be a factor that governs the consistency of the subdural collection. Hemorrhage from a traumatically opened vein that traverses the subdural space without injury of the brain itself should result in a different type of subdural collection from that following contusion-laceration

of the cortical surface with the attendant opening of the subarachnoid space. In the latter instance the blood in the subdural space would be diluted with cerebrospinal fluid, thereby affecting the clot formation. The laceration of the brain heals, leaving a small zone of cortical atrophy covered with thickened pia-arachnoid. All stages of such healing processes have been observed, from the fresh, reddish-brown subpial hemorrhage surrounding a zone of brain laceration to the milky white pia-arachnoid adherent to the inner membrane of a subdural collection. One cannot infer from the appearance of the cortical surface at operation, the extent of the pathologic process that had been present.

There were 136 of the 143 patients in this series who had examinations of the skull roentgenologically or at autopsy. Sixty had fractures of the skull, and in 76 cases no fracture was demonstrable. As previously stated, there are rare instances in which a fracture of the skull has resulted in a laceration of a meningeal artery and the dura, thereby opening an avenue for the arterial blood to gain entrance to the subdural space. There were six cases in this series that had both extradural and subdural hematomata. The extradural hematomata were proportionately small, consequently these cases are included in the subdural series. In three instances the clots were on the same side, in two cases the subdural collection was on the opposite side to the epidural hemorrhage, and in one there were bilateral, small extradural with unilateral subdural hematomata. Subdural hemorrhage is an occasional finding in association with compound depressed fracture of the skull, but this type of case has not been included in this series. In practically all instances of subdural hematoma the presence or absence of fracture of the skull has very little related significance. From the evidence at hand one may reasonably state that subdural hematoma is a lesion resulting from trauma to the head and is frequently found with any or all of the following: Lacerations of the scalp; hematomata beneath the scalp; all varieties of fracture of the skull; extradural hemorrhage; contusion-laceration of the cerebral cortex; intracerebral blood clots; and intraventricular hemorrhage.

The patients who survive the acute phase of the craniocerebral insult make up the group that come to operation or autopsy at a later date and are usually designated as chronic subdural hematoma. In these cases the finding of obvious edema of the brain adjacent to a long-standing subdural collection (Fig. 4), encased by a fibrotic membrane, strongly suggests that the symptoms of a space-occupying intracranial lesion, appearing after a considerable interval has elapsed following the trauma, are due to this edema of the brain rather than increase in the fluid content of the subdural collection.

Symptoms, Physical Signs and Diagnosis.—The majority of patients harboring a subdural hematoma present evidence of a recent trauma to the head or later, when seeking medical counsel after an interval of relative freedom from symptoms following the injury, admit a previous accident. There are a few from whom no history of trauma can be obtained either by direct questioning or from the relatives. In this series there were two cases in which

trauma could be excluded as an etiologic factor. In 101 cases the injury to the head resulted in immediate unconsciousness; 22 patients were not rendered unconscious by the blow; and in the remaining 18 cases the exact state of consciousness after the trauma could not be ascertained. Of the 101 instances in which there was an unquestionable period of unconsciousness immediately following the trauma, there was an interval of lucidity before secondary stupor supervened or before operative intervention in 52. Thirty-six of the remaining 42 patients were in stupor or coma from the time of injury until operation was performed or until death occurred. Eighteen of these 36 patients were operated upon, with only two recoveries. Evidence of recent consumption of alcohol was present in 68 cases. Convulsions occurred in ten cases. Headache, vomiting, double vision, weakness or numbness of the extremities on one side, and stiffness of the neck were recorded in various combinations but these could be considered of value only when present during the late or chronic phase of the disorder. Vomiting was noted in 57 cases and headache in 79. The patients who were initially unconscious following the trauma and subsequently became oriented, presented one quite characteristic type of behavior. Noticeably during the so-called lucid interval, many were observed to lapse from a cooperative and alert state into a stupor within a period of 20 to 30 minutes, shortly to be followed by a return to the previous conscious level. Several such cycles were apt to occur in a single day. While this sequence of events may be observed in association with conditions other than subdural hematoma in combination with obvious trauma, it suggests the presence of a "compression lesion" rather than an intrinsic pathologic process of the brain. After three weeks to a month, symptoms are commonly more clearly defined, although in some instances they appear to be entirely unrelated to a trivial accident that has long since been forgotten. More often the persistence of paroxysmal headache will link the illness to a previous trauma of the head. The concomitant symptoms that occur during the chronic phase are usually those associated with any space-occupying lesion of the intracranial cavity.

The important physical findings to be given consideration during the acute phase of subdural hematoma are usually limited to the obvious external evidence of trauma, the state of consciousness, the condition of the pupils, the appearance of the optic fundi, gross alterations in facial movements, gross weakness of an extremity or of the extremities on one side, and the status of the superficial and deep reflexes. The state of consciousness is usually such that precision in neurologic examination is impossible. The abnormal recorded findings that were reviewed in this series are difficult of evaluation, since many no doubt were the result of intrinsic brain damage (petechial hemorrhages, edema, laceration, *etc.*). Repeated examinations are important, for changes in physical signs are regarded as being of more significance than the information obtained at any one examination. The age of the patients in the present series ranged between 14 months and 80 years. Most were in the fifth decade of life. The pupils were recorded as being equal in 59 and

unequal in 72 cases. Only 12 of the latter showed an unequivocal wide dilatation of the pupil on the same side as the blood clot and in one of these cases there was a bilateral subdural hematoma. In this instance the larger clot was found at operation to be on the same side as the dilated pupil. The pupil was larger on the same side as the lesion in 32 cases but could not be considered as "dilated." Twenty-six cases had the larger pupil on the contralateral side with respect to the hematoma. There were two examples of bilateral subdural hematoma in which the pupil was observed to be larger first on one side and then on the other (shifting dilatation of the pupil). The state of the pupil in the remaining 12 cases in this series was not recorded with sufficient detail to permit accurate classification. Papilledema was recorded in 50 of the 143 cases. This included all grades, from slight blurring of the superior nasal border of the optic disk to the most marked degree. Here again, one could not be sure that the presence of the blood clot in the subdural space caused the edema about the optic disk, or whether the clot in combination with diffuse brain changes dependent on the trauma resulted in increased intracranial tension. Nuchal rigidity was observed in 57 cases. The most striking rigidity of the neck has been noted in association with blood in the cerebrospinal fluid. This rigidity has frequently been interpreted as representing the reaction to the blood in the spinal subarachnoid space; however, there are many instances in which such rigidity has been observed following uncomplicated trauma to the head, while the cerebrospinal fluid was found to be normal. Unilateral paresis of the face, central type, was observed in 80 cases. In 22 cases the facial weakness was on the same side as the subdural hematoma and in 58 instances it was contralateral. Facial paresis without demonstrable weakness of the homolateral extremities was recorded in 15 cases. A hemiparesis implicating the motor power of the face and upper and lower extremities of one side was recorded in 61 cases. The motor weakness was contralateral to the subdural lesion in 42 cases and homolateral in 19. The abdominal reflexes were absent bilaterally in 99 cases and not present on the side of the motor weakness in 20. Babinski's sign was present bilaterally in 67 cases and unilaterally (side of motor weakness) in 25. The scanty information derived from the sensory examinations was unreliable. Incontinence of urine was recorded in 108 of the 143 cases. Bradycardia (pulse rate below 70 per minute) was present in 63 cases. The cerebrospinal fluid pressure was elevated in 79 cases (above 15 Mm.Hg.), the fluid contaminated with blood in 80 cases, xanthochromic in 30 cases and clear and colorless in 19.

A clinical diagnosis of subdural hematoma can be made during the acute phase of the disorder with a fair degree of certainty provided only a moderate grade of trauma has resulted in a loss of consciousness for 48 to 72 hours, followed by a few days of lucidity and then a recurrence of the stupor. There are a few cases in which only a momentary alteration of the conscious state, the consequence of the initial insult, is succeeded by normal activity for a few hours to several days and then by progressively increasing drowsiness. This

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exploratory bur opening in the skull or by air studies. A third group
acute cases includes those that remain in coma from the time of injury
the blood clot is removed at operation or until death. The lesion most
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of the brain is frequently associated with a subdural hematoma, the
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Roentgen-ray Examination.—Plain roentgenographic studies of the head are of distinct value in many cases. The presence or absence of a fracture of the skull aids little in the diagnosis of subdural hematoma. A demonstrable shift from the midline position of a calcified pineal body is confirmatory evidence that a space-occupying lesion is present and indicates the side of the lesion. If trauma to the head has been sustained it is logical to assume that the space-occupying mass is a blood clot. A mild shift of the pineal may occur in simple edema of one cerebral hemisphere following trauma and the same may be said of a shift of an air-filled ventricular system. The most important application of the roentgenologic study of the head with particular reference to the diagnosis of subdural hematoma is made after the introduction of air into the ventricular system. One of two methods may be utilized—encephalography or ventriculography. The former method of examination was employed preoperatively in 45 cases in this series and the latter in 27 instances. In 19 of the 45 encephalograms, air failed to enter the ventricular

system, although in several of these failures air was present in the cerebral subarachnoid space and the side of the lesion could be determined by the position of the falx cerebri. The injection of air by way of the lumbar subarachnoid space seemed to have been a factor in the subsequent death of one patient.

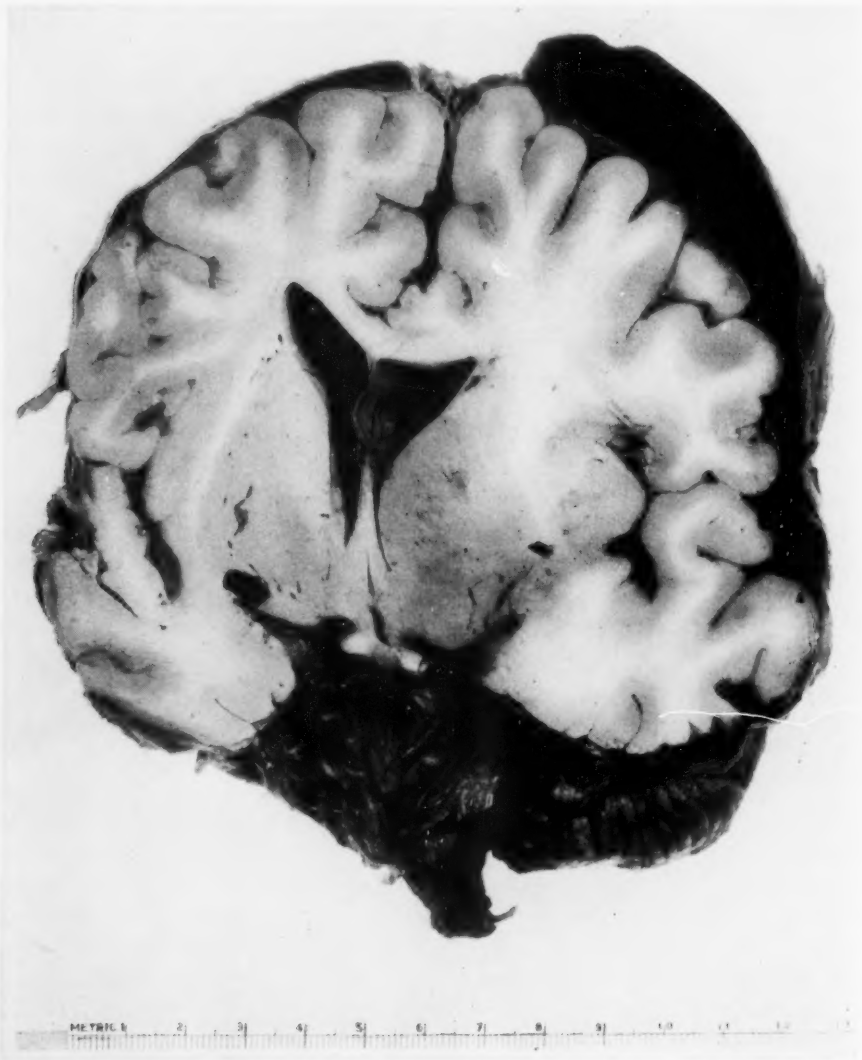

FIG. 4.—Subdural hematoma of three months' duration. Note the edema implicating the capsular zone on the side of the hematoma.

The 27 ventriculographic studies clearly indicated the side and position of the lesion. The air studies carried out on patients in the acute phase of their illness gave evidence that a space-occupying mass was present and suggested a diffuse compression of one or the other cerebral hemisphere. The sharply defined defect in the ventricle produced by a chronic circumscribed hema-

toma resembled the roentgenographic findings associated with meningiomata. Eight verified cases of edema of a cerebral hemisphere, not included in this series, showed a shift of the ventricular system in every respect similar to that produced by subdural hematoma. During the past two years there has been a tendency toward preference for the direct introduction of air into the ventricular system, although spinal subarachnoid injection of 40 to 50 cc. of air has been employed in selected cases without deleterious effects.

Treatment.—There is but one form of treatment for subdural hematoma: surgical removal of the blood clot. Multiplicity of intracranial lesions dependent on the injury that initiated the subdural bleeding are frequently present. Removal of the subdural collection without continued supportive treatment as has been outlined elsewhere[13] will frequently be followed by a fatal outcome. As would be expected, if the patient can weather the storm during the first three or four days following the injury, the chances for recovery are greatly enhanced. Table III illustrates this very clearly. On the other hand, some patients are saved by an operation performed during the first three or four days after the traumatic insult to the brain. Table IV illustrates that it is not necessarily the size of the clot that determines the outcome.

TABLE III

Elapsed Time between Injury and Operation	Total	Recovered	Died	Mortality
2 to 24 hours	24	4	20	83.3%
1 to 7 days	38	23	15	39.4%
7 to 14 days	17	11	6	35.3%
14 to 21 days	19	16	3	15.7%
21 to 28 days	5	4	1*	20.0%
Over 28 days	8	8	0	

* Patient reported in text to have had a subdural collection associated with multiple metastatic foci of infection in the brain.

TABLE IV

Weights of Clots	Total	Operated and Recovered
10 to 25 Gm.	2	1
25 to 50 Gm.	5	3
50 to 75 Gm.	54	24
75 to 100 Gm.	34	7
100 to 150 Gm.	35	24
Over 150 Gm.	13	7

Knowledge derived from statistics, however carefully compiled, has certain limitations in its application. Repeated review of our results in the treatment of patients with subdural hematomata has from time to time modified the method of managing these patients. No absolute standard is adhered to, since a considerable flexibility of thought is desirable and advantageous in the treatment of each individual. Conservative treatment of patients immediately after a craniocerebral injury is indicated except in those instances in which a mild to moderate degree head injury associated with unconscious-

ness is followed by a lucid interval and then within a relatively short period of time by stupor. If after a 10- to 20-hour period of observation, the vital signs and the conscious state indicate that they are not doing well on a conservative therapy, and the physical signs suggest the probability of a complicating intracranial hemorrhage, operation is performed. With the patient in the face-down position on a cerebellar outrigger, bilateral bur openings are made in the skull slightly more distant from the midline and somewhat anterior to the conventional position used for ventricular puncture. If upon opening the dura a fluid subdural collection is encountered, this is evacuated. If the hematoma is solid a small bone flap is turned down and the clot removed. In the event that no subdural collection is disclosed through the bur holes but the clinical findings suggest the presence of an intracranial hemorrhage, ventriculographic studies are performed. If the roentgenologic findings indicate the presence of a blood clot the patient is returned to the operating room for its removal. This general outline of therapy applicable to many patients during the first four or five days following injury (and in our opinion frequently mandatory) is not pursued if it is estimated by the clinical course that they may survive the acute phase of the injury. The mortality is high during this period but it can be somewhat reduced by judiciously applied surgery. Many of the subdural collections are solid clots during the first few days, and consequently require a bone flap for proper removal. After eight to 12 days some of these become liquefied and can be evacuated through a bur hole in the skull. Once a patient has recovered from the immediate effects of a brain injury, yet, as judged by the clinical course, a subdural hematoma is present, operation should not be unduly delayed. It is far better to perform a ventriculogram in the absence of a blood clot than to permit a patient with a subdural collection to lapse into a secondary stupor, at which time the removal of the clot may be of no avail.

The chronic phase of subdural hematoma presents no particular therapeutic problem unless the patient is admitted to the hospital in coma, requiring prompt attention. If at operation the bur opening is made over the hematoma, the thick outer membrane may be incised and the contents of the hematoma, if liquid, evacuated. Failure to disclose the lesion beneath the conventional posterior site for bur openings made for ventricular puncture should be followed by air studies and a small flap turned down over the involved area. Upon exposing the outer membrane it is not necessary to cut an opening more than 3 to 4 cm. in diameter to completely evacuate the contents of the subdural collection. The outer membrane of a chronic lesion is quite vascular; therefore, the incision through this structure should be made with the electrocoagulating unit. Absolute hemostasis is essential. The relatively fresh blood clots offer few technical difficulties but the chronic, fibrosed lesions may be intimately attached to adjacent blood vessels. The latter type should be removed piecemeal as is required in some meningiomata.

Discussion.—The frequency of occurrence of subdural hematoma in this five-year study, 143 in 10,265 cases of cerebral injuries, is much less than

that reported by other authors.[9, 10, 14] Several reasons present themselves in explanation of this discrepancy. All instances of laceration of the brain with a layer of blood clot over the site of laceration and some in which there was a small amount of subdural blood clot about the laceration have been excluded

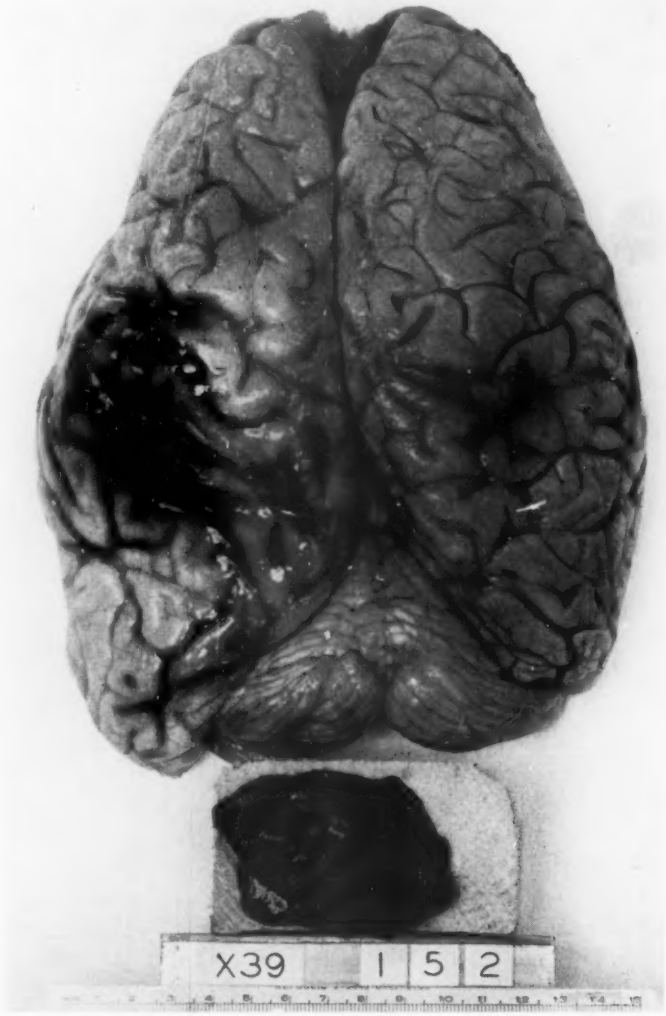

FIG. 5.—Small subdural hematoma taken from the zone of contusion-laceration as shown.

from this series (Fig. 5). It is our belief that most patients suffering such pathologic states recover, and in support of this contention two autopsy specimens recently examined will be referred to as examples of what may happen to similar lesions in others. The story of a previous accident was obtained in both cases, one sustaining a head injury about one year previously, and the other approximately eight months before the fatal issue. Both subjects

had died as a result of a second craniocerebral injury, and at autopsy a fresh blood clot was disclosed over one cerebral hemisphere, whereas on the other side a tough, fibrosed homogeneous structure 3 to 4 mm. in maximum thickness occupied the subdural space. During the periods between the accidents one patient was said to have been free of symptoms and the other patient had recurrent episodes of headache although able to perform manual labor. There was ample evidence in both specimens to show that the older subdural collections followed lacerations of the temporal lobes. Furthermore, many small clots of the character illustrated by Figure 5 have been exposed at operation and the lesions classified as cerebral contusion-laceration of the brain. Approximately one-half of the 10,265 patients with head injuries presented evidence of mild brain trauma, and it is possible that a few with unrecognized subdural collections of one character or another were discharged free of symptoms but subsequently admitted to other hospitals. Two instances in this category are definitely known. No doubt many of the patients with chronic subdural hematomata that have been reported in the literature are individuals who have largely recovered from a mild head injury, and because of an exacerbation of symptoms have eventually been directed to institutions where cranial surgery is being performed.

The position of a subdural hematoma does not necessarily signify the region from which the bleeding occurred. Many autopsy specimens have been examined which disclosed a laceration of the temporal and/or the orbital surface of the frontal lobe associated with a sizable hematoma in the parasagittal position (cases similar to that illustrated in Figure 2), yet no injury to the crossing veins in this region could be demonstrated nor could the slightest attachment of the blood clot to the underlying pia-arachnoid be shown. Most chronic subdural hematomata are located in the parasagittal region, and because of this it appears that many authors have expressed the opinion that the blood vessels here are frequently opened traumatically. In view of the findings at autopsy in many fresh specimens, it seems probable that a subdural collection may eventually assume a position somewhat distant from the original source of bleeding. Secondary hemorrhages are reported to occur from the outer membrane of a long-standing subdural collection. We have never recognized this lesion in any of the material examined. There can no longer be any question that the pathologic lesion, brain trauma complicated by a subdural hematoma, undergoes changes that result in sufficient increase in volume on the affected side to cause a progressive shift of the cerebral ventricular system (Fig. 6). This has been clearly demonstrated by series of air studies in several instances. While it may be held that this progressive displacement of the ventricular system may be caused by edema of the brain adjacent to the blood clot, it has been observed that patients in whom this increasing shift has been demonstrated may be showing clinical improvement. It seems unlikely that recovery from stupor and improvement in motor activity of a previously paretic extremity would go hand-in-hand with an increasing displacement of the ventricular system away from the lesion if the

increment were due to brain edema. Another interesting fact disclosed by repeated air studies on patients with subdural hematoma is worthy of comment. The initial air study always shows, as would be expected, a relative narrowing of the lateral ventricle on the side of the lesion and mild to moderate dilatation of the contralateral ventricle. Studies carried out after removal of the blood clot demonstrate that this inequality in size of the lateral ventricles remains, although both frequently become moderately dilated (Fig. 6C). This observation has been verified as late as one year after the evacuation of a subdural collection.

There were two examples of hematoma located in the subdural space of the posterior fossa included in this series and, in addition, we have observed

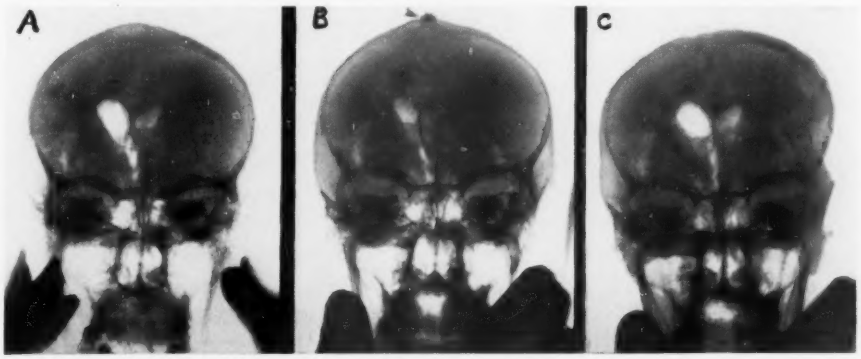

FIG. 6.—A series of roentgenograms representing changes in the size of a subdural collection. (A) Anteroposterior view showing a mild displacement of the ventricular system on the seventh day following injury. (B) The same view at a second examination 18 days later. Note the advance in the ventricular shift. (C) Third examination 10 days after surgical removal of an hematoma.

three other cases prior to 1934. A clinical diagnosis of this lesion has never been made; in fact, in only one of these five cases was this diagnosis entertained as a probability. Although the ventriculographic findings in one instance disclosed a mild symmetric dilatation of the lateral ventricles that were centrally placed, the deductions, from all the evidence at hand, did not lead to the correct conclusion. The clinical diagnosis of posterior fossa neoplasm was made in three children, all of whom showed only two abnormal physical findings: high degree of choked disk and unsteadiness of gait. A fluid subdural hematoma was disclosed in each instance upon making bur openings for ventriculographic studies. Spinal puncture had been performed in each of these three cases and the fluid found to be clear and colorless. As previously stated, there were all together 22 cases in the series that showed clear cerebrospinal fluid, although many had spinal punctures performed on several occasions during the acute phase of the illness. Furthermore, the pressure of the cerebrospinal fluid may be high, low or normal. Several verified instances of large subdural collections have been found to show very low cerebrospinal fluid pressures as measured in the lumbar thecal sac. In some cases this low intracranial pressure was substantiated at operation by the fact that upon opening the outer membrane of a subdural collection through a small

cranial opening only a few cubic centimeters of liquid blood were discharged. After washing out 100 to 150 cc. of old fluid blood, some patients regain consciousness while still on the operating table. Upon inspecting the brain through the cranial opening, the cortical surface would commonly be found at a level 2 to 3 cm. below the under surface of the dura. We have been unable to interpret these findings in terms of the generally accepted concept regarding the relation between intracranial pressure and altered conscious states. Not infrequently under the above conditions, a patient will remain conscious for 18 to 24 hours after removal of a hematoma and then become stuporous again. After dehydration therapy an uneventful recovery usually ensues. The secondary stupor has been thought to represent cerebral edema depending upon postoperative expansion of the compressed brain. It has also been observed, especially in older patients, that a failure of the brain to resume its normal contour shortly after the removal of the blood clot is an unfavorable prognostic sign.

The adage that a subdural collection of blood (fluid, clotted or a mixture of the two) should be removed once the diagnosis has been established, is fallacious in our experience. A study of Table III clearly indicates the high mortality attending operations performed during the first 24 hours following injury. The fatalities could not be considered attributable to the operation *per se* in view of the severe brain injury found to be present. In each patient operated upon within 24 hours after injury, it was believed from the clinical course that death would certainly ensue unless some measure were instituted to alter the downhill trend of events. We have concluded from experiences during the past nine years that a greater percentage of the severely injured who have as a complication a subdural hematoma will survive if the hematoma is removed, even during the first 24 hours, than through the institution of conservative measures during the early acute stage and operation at a later date. The records indicate further that those not so seriously injured and who survive for two weeks or longer have an excellent chance for recovery following the removal of the hematoma. Operations performed during the chronic stage should carry a very low mortality. The 32 patients (Table V) that were not operated upon are all examples of severely injured

TABLE V

Patients operated	111		
Recovered	66		
Postoperative deaths	45	Operative mortality	40.9%
Not operated	32	Mortality	100.0%
Total deaths	77	Total mortality	53.8%
Autopsies	68	Autopsies	88.3%

who died shortly after admission and in whom the lesion was verified at autopsy. A subdural hematoma was suspected to be present in many of these on the basis of clinical findings, but in each instance the general physical states were such that operation was thought to be inadvisable. Approxi-

mately one-half of those who recovered have been followed in the Out-Patient Department. The sequelae (headache, dizziness, convulsion, *etc.*) that have occurred among these have been in about the same proportion as that encountered in any group of patients who have sustained moderate to severe trauma of the brain. Three patients were committed to psychiatric care but these recovered, on the average, in one year's time and were discharged from the hospital.

CONCLUSIONS

The important features relative to the etiology, pathogenesis, diagnosis and treatment of subdural hematoma, as revealed by a study of 143 cases, have been presented. This series of cases encountered in a five-year period (July 1, 1934, to July 1, 1939) represents all patients in whom this lesion was verified either by operation or at autopsy. The data derived from the study indicate that in most instances subdural hemorrhage arises from a laceration of a cortical vessel resulting from trauma to the head. Frequently there is a concomitant traumatic lesion of the brain, the effects of which cannot be delineated from those produced by the blood clot in the subdural space. Some patients are obviously so seriously injured that no benefit can be derived from surgical measures. Many others surviving the cerebral insult for a few hours to a day, and presenting clinical evidence that a fatal outcome may be expected, are not improved by the removal of the subdural hematoma. A few so injured will recover and the recovery can, in the main, be attributed to the prompt removal of the blood clot. The clinical course of some patients who survive the acute phase of a cerebral insult accompanied by a subdural hematoma suggests that an increase in the size of the subdural collection may occur. Encephalographic studies repeated at varying intervals in instances in which a subdural collection was shown to be present, have demonstrated a progressive shift of the ventricular system away from the lesion. This advancing shift of the ventricles seems to result from an increase in the volume of the subdural collection. Whenever doubt exists regarding the diagnosis of subdural hematoma, air studies may be utilized to great advantage in clarifying the issue. Surgical removal of a subdural collection is the only therapy. If feasible, operation should be deferred until the acute effects of the injury of the intracranial structures have moderated.

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DISCUSSION.—DR. CHARLES ELSBERG (New York) asked if he had understood Doctor Browder to say that he believed the symptoms of increased pressure that occur from subdural hematomata are due not to added fluid but rather to edema of the brain. If chronic subdural hematoma is a continuation of the acute stage, how is it that a slowly advancing papilledema, as evidence of increased intracranial pressure, occurs in patients who did not have evidence of increased pressure for many months? When one operates upon such a patient and evacuates the hematoma, it is lined both on the undersurface of the dura and against the pia arachnoid by a soft membrane. If one splits that membrane one very often sees the brain gradually bulge to only a slight extent. How could edema of the brain explain a condition of that kind?

Much has been said regarding the treatment of subdural hematoma merely by the making of trephine openings and the evacuation of fluid with blood clot and organized material. Doctor Elsberg's experience, during the course of many years, has been to find that while such a procedure would relieve conditions somewhat, the real relief of pressure upon the brain is obtained only when the inner membrane has been split, and usually a large amount of it excised.

Was not Doctor Browder speaking of an acute process with injury to the brain and secondary edema to the brain rather than of a chronic subdural hematoma?

DR. ERNEST SACHS (Washington University Medical School, St. Louis, Mo.) said that if he understood Doctor Browder clearly, he was inclined to believe that chronic subdural hematoma followed acute subdural hematoma, and that the increase of a subdural hematoma, an undoubted occurrence, is not due to the accumulation of fluid as believed by many and proven rather strikingly some years ago by Gardner in Cleveland. Doctor Sachs cited a case which in his opinion indicated beyond doubt that chronic subdural hematomata do increase in size. Some years ago he saw a patient who gave the history that while tobogganing with his wife they were both thrown off. His wife, although unconscious, proved to be only slightly injured. He, however, after recovering apparently completely, noted, five months later, symptoms of unilateral paresis, until finally he had the typical picture of an intracranial expanding lesion. At operation, he had a huge subdural hematoma. Unquestionably, he sustained a slight trauma at the time his wife was injured and may have had a clot such as Doctor Browder spoke of at the time of the injury, but something happened in the course of five months to give that man

symptoms. The thing that happened was that the subdural hematoma increased in size, and did so because the sac contained fluid. Where did that fluid come from? Certainly, one cannot suppose that a vessel oozed for five months. The contents of a subdural hematoma is fluid, as Gardner showed in one case where he completely removed the membrane and obtained fluid, proving that fluid did enter the membrane. This and similar experiences have convinced Doctor Sachs that chronic subdural hematomata do increase in size from the time they first form.

Another point that caused Doctor Sachs to wonder, was whether the kind of subdural hematomata that Doctor Browder spoke of and chronic subdural hematomata are the same sort of thing, just in a different stage, because, in the experience of most surgeons, chronic subdural hematomata very frequently occur bilaterally. In Doctor Browder's series of 143 cases he had only ten bilateral hematomata. That is certainly very different from the experience of most people with chronic hematomata.

A third point with which Doctor Sachs could not quite agree was with regard to treatment. During the early days he believed that the thing to do was to turn down a flap and remove the subdural hematoma, but in recent years, as cases have been seen increasingly, he has been completely won over to the other point of view, namely, that of his associate, Doctor Furlow. First bur-openings are made on each side to determine whether there is an hematoma, and, if so, it is washed out. As a rule, apparently, if the contents of the hematoma are thus thoroughly evacuated, the membrane takes care of itself. If, after trying this simpler procedure, the patient is not completely relieved, a bone flap is turned. Doctor Furlow reviewed all of his and Doctor Sachs' subdural hematomata some years ago, and found that most of them had been relieved by the simpler procedure. Only rarely was it necessary to turn down a flap.

Dr. Ira Cohen (New York) said that if one accepts as a definition of hematoma a collection of blood or clots in the tissues of the body or closed spaces, then it would seem justifiable to group the acute collections of blood in the subdural space and the chronic subdural hematomata in one discussion. It may have the advantage of calling to mind or emphasizing that such a collection or a chronic subdural hematoma—space-occupying lesions in the subdural space—are surgical problems and should be handled surgically. Doctor Browder more than hinted at the fact that although they are all collections of blood in the subdural space, clinically there is a real difference between a so-called acute hematoma—which Doctor Cohen prefers to call a collection of blood in the subdural space—and a chronic hematoma. There is a difference clinically, because in the acute cases there is no question of the close connection between the trauma and the lesion. Moreover, in the acute case the differential diagnosis, as brought out by Doctor Browder, between the responsibility of the hematoma, of the underlying brain lesion, or the brain edema, for the dominant part of the picture, is not an easy one. On the other hand, the chronic subdural hematoma has a different clinical picture and these hematomata present themselves, in Doctor Cohen's experience, in four groups:

In the first group, the patients refer to a trauma received some weeks or months previously, a slight trauma (in some cases so slight as that of a grandmother who has been hit on the bridge of the nose by a grandchild held in her lap) in contradistinction to the severe trauma in the acute collections. These patients connect their symptoms—their headache, or whatever their complaints are—with this remote trauma, and one realizes the connection.

In the second group, the patient often enters the hospital in stupor or semi-

stupor, most often without a history of trauma, and for the time being is regarded as a cerebrovascular accident.

In the third group, one encounters a picture practically akin to that of a brain tumor. A history of trauma is sometimes obtained preoperatively, but usually not until after the operation, and such patients are regarded as having brain tumors until the operation, or in an attempt at air studies an hematoma is found in the subdural space.

The fourth group is practically made up of patients with purely mental symptoms, without a history of trauma. The patient comes in with memory or personality changes, and with almost no sign of organic disease. Here again, an hematoma is diagnosed or thought of only when bur-holes are made for air studies.

As to increase in size of the hematoma, especially chronic hematomas, Doctor Browder did say that he felt that Doctor Gardner's theory is correct. He also referred to Dandy's remarks on edema being the cause of the presenting symptoms. Doctor Cohen said he had no doubt that the hematoma increases in size by osmosis, as Gardner stated, but he also felt that in many cases the final symptoms are due to edema. In one group of hematomata, at operation, the hematoma may be removed and the brain not expand. Certainly, the brain is not the seat of edema in such an instance. On the other hand, when one punctures the outer membrane of hematomata the fluid will squirt out of the needle and Doctor Cohen felt that such a case is one where the pressure is exerted by the intrahematoma pressure. In another group, when the needle is introduced the fluid drops out or has to be aspirated. It is hard to believe that the semistupor of such a patient can be caused by the intrahematoma pressure. Therefore, in those cases one may very well be dealing with an edema of the brain.

Dr. E. Jefferson Browder (Brooklyn) thanked Doctor Cohen for having clarified some of the statements made in the presentation. The illustrative encephalograms that were presented, showing progressive increase in the shift of the ventricular system associated with subdural hematoma, were typical of the findings by repeated air studies in seven other cases. Whether or not this progressive shift of the ventricles is the result of edema of the brain or an increase in the size of the subdural collection, cannot be stated with certainty. All subdural collections are not in a fluid state. Some are encountered as solid as a meningioma. It seems difficult to postulate any theoretical explanation that would meet all requirements in instances in which such a solid hematoma is present for several months without producing symptoms and then, over a period of a few days, causes drowsiness followed by stupor and hemiplegia. Doctor Browder said he could not believe that an organized mass of this type can be augmented by an influx of fluid. An attempt was made, in his presentation, to show that there are many types of subdural collections. On, say the fifth day after injury, one may encounter solid clots; orange-tinted fluid; black, tarry fluid blood; thin, dark brown substance, *etc.* Possibly a dozen different types have been observed. It seems that many of the fluid hematomata increase in size by osmosis, but the organized clots are seldom if ever altered in this manner.

Doctor Elsberg discussed the question of operative approach and method of evacuation of the subdural collections. In some cases the fluid hematoma may be evacuated through one, or, if desired, several bur-holes in the skull, whereas solid clots, as well as some fluid collections, require a small bone flap. At the present time Doctor Browder is using small bone flaps much more frequently than formerly and is in entire agreement with Doctor Elsberg's

statement that subdural hematomata can be more adequately handled through a large cranial opening than by washing out the collection through a bur-opening.

Chronic subdural hematomata, in Doctor Browder's opinion, represent the late phase of the acute hematoma. He has encountered subdural collections from two hours to two years following injury. Those included in the present study were disclosed at operation or at autopsy within three months of injury, most of them within one month.

Doctor Browder said that he had presented the subject as objectively as possible, an attempt being made to portray the problem of head injury complicated by subdural hematoma as it has presented itself in a large city hospital. The 143 cases mentioned occurred in 10,265 admissions, or approximately 2 per cent. Munro has reported a much higher percentage from the Boston City Hospital. In Doctor Browder's series, all instances of laceration of the brain with a thin layer of blood over the site of laceration were excluded, although these are, in the true sense, subdural hematomata.

EXTRADURAL HEMORRHAGE

A STUDY OF FORTY-FOUR CASES

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EXTRADURAL HEMORRHAGE is a dramatic and theoretically well-recognized traumatic lesion of the cranium. Much has been written about it in both early and recent literature. However, after reviewing the reported cases, it has been our feeling that we need make no excuse for bringing the problem forward again. Most of the teaching in medical schools and in the standard text-books of general surgery is misleading or, at the best, cursory. The so-called classical picture, with its initial unconsciousness, lucid interval and secondary period of unconsciousness plus clear spinal fluid under elevated pressure, is so rare as to be the exception rather than the rule. This classical picture has been the more firmly entrenched because some authorities, both neurosurgical and pathologic, have insisted that the bleeding is always arterial. We feel definitely that, in a certain portion of the cases, the source of the extradural bleeding is venous in origin. These facts have also been recognized by other authors.

Our series of 44 cases is somewhat larger than any group heretofore reported from one clinic. It seems to us that certain very definite conclusions may be drawn from an analysis of the results of the fairly standardized method of treatment that we have used over a number of years.

Hippocrates recommended that the cranium be perforated when injuries might be followed by serious consequences such as the extravasation of blood. Celsus advised waiting until untoward symptoms appeared before operating. Since then, many articles have been written on this subject. The more important authors are listed in the references.[1–8] The classical work on extradural hemorrhage, however, is that of W. H. A. Jacobson,[9] written in 1885. Little has been added to the picture since this appeared. He thoroughly analyzed 70 cases; 27 from Guy's Hospital and 43 from the English literature. Any report on this subject must refer frequently to his excellent clinical descriptions. Since that time, several papers reporting the clinical findings and treatment of small groups of cases have appeared annually. In 1938, Pringle[10] published what appeared to be another large series. This was a review of 71 cases of traumatic meningeal hemorrhage. However, in analyzing these cases only 23 can be considered as true extradural hemorrhages, the others being subdural hematomata.

In reviewing the literature, several things stand out: First, many of the early authors were aware of the rarity of the so-called classical picture. Second, not a few of the men writing on this subject realized the probability of venous bleeding as a source of the clot in some of their cases. Finally, there

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has been no large series of cases reported previously by a single clinic in which a well-standardized method of treatment was used.

Incidence.—Extradural hemorrhages are by no means a common complication of a blow on the head. This is illustrated by the fact that in over 1,200 head injuries treated on the Neurosurgical Service of the Boston City Hospital from 1932–1939, only 44 or about 3 per cent showed extradural hematomata. LeCount and Apfelbach[11] report a study of 504 cases that had fractures of the skull discovered at autopsy. In their series, 19 per cent showed extradural hemorrhage which produced appreciable compression of the brain. Moody[12] studied 908 cases of proven fracture of the skull and found 100 instances of extradural hemorrhage (9 per cent).

Etiology and Pathogenesis.—The immediate cause of a traumatic extradural hematoma is, of course, a blow on the head, which may or may not leave external evidence of violence.

The common assumption is that extradural hematomata are always caused by a rupture of the middle meningeal artery or one of its larger branches. This belief has been inculcated by certain authoritative statements to the effect that arterial, and only arterial, bleeding was capable of stripping the dura from the bone. We feel sure that in certain instances the source of the bleeding is venous, either from a great venous sinus or one of the smaller veins. Wood Jones,[13, 14] in two excellent papers, has proven, beyond a doubt, that the grooves in the skull usually believed to be due to the pulsating arteries, are for the most part produced by the veins that accompany these arteries. His convincing discussion will not be elaborated upon here. However, in 1911, anatomists had entirely accepted his findings, although most surgeons are still not aware of this anatomic fact. Wood Jones not only made purely anatomic studies but by injection methods, in three cases in which death had occurred from massive extradural hemorrhage, proved that the venous sinuses around the arteries were torn while the arteries themselves were intact. Verbrugghen[15] has recently reviewed and reiterated the findings of Wood Jones and has reproduced one of the latter's graphic illustrations. In the majority of cases where the bleeding point was definitely observed it has been arterial, but in a smaller number the bleeding was seen to come from one of the great venous sinuses or a tributary vein. LeCount and Apfelbach say that in their series the bleeding was attributable to the anterior branch of the middle meningeal artery in 49 cases and to the posterior branch in 44 cases. They also found three instances in which the bleeding was from a laceration of the superior longitudinal sinus, and eight in which the bleeding was considered to have originated from the lateral sinus. Erichson,[7] Jacobson,[9] and Verbrugghen[15] also record instances of venous bleeding as the source of extradural clots. McKenzie[16] stresses the fact that the bleeding is not always arterial in origin; and speaks of three cerebellar clots. One of these was found at operation. They were caused by torn lateral sinuses. Our series, likewise, includes one cerebellar extradural hematoma. This was found at autopsy, the source of the bleeding being from the lateral sinus.

In our 44 cases, the source of the bleeding was definitely mentioned in

37 instances (84 per cent). In 26 (70 per cent) the bleeding was described as from a portion of the middle meningeal artery. However, it must be mentioned that if the contentions of Wood Jones and others are correct, it is probable that even in certain cases in which the middle meningeal artery was assumed to have been torn, the bleeding, especially if it had not been furious, might have been from the surrounding venous channels. In the 26 cases in which the middle meningeal was said to have been torn, there was a marked variation in the description of the difficulty of controlling the bleeding, and also in the length of the lucid interval, as compared to those with a known venous source for the hemorrhage.

In ten cases in our series there was definite mention of a venous, and only a venous, origin for the bleeding. In seven, the source was demonstrated to have been one of the lateral sinuses. In three, the bleeding was found to have originated from a rent in the sagittal sinus or from one of its large tributary veins.

Thus, a review of the literature and a study of our own experience indicates that the source of extradural bleeding is usually but not always a rupture of the middle meningeal artery or one of its branches. Venous bleeding as a source for the clot has been neglected in discussions of the etiology and pathogenesis of extradural hematoma. To thoroughly understand the clinical and therapeutic problems related to extradural hemorrhages one must be aware of this latter possibility.

TABLE I

SUMMARY OF SYMPTOMS IN 44 CASES OF EXTRADURAL HEMORRHAGE

Finding	Times Mentioned	Contra-lateral	Ipsilateral	Bilateral	Remarks
Hemiplegia	25	22	3	—	
Facial weakness	13	11	2	—	2 ipsilateral, peripheral type
Babinski	34	10	1	23	
Spasticity or rigidity of extremities	10	4	3	3	3 spastic quadriplegias
Abdominal reflexes	9	Absent 5	Absent 1	Absent 3	
Deep tendon reflexes	18	Increased 12	Increased 1		Absent in 5
Papilledema*	4			4	Interval between injury and observation: 4, 5, 7, and 11 days
Sensory changes	2	2			
Nuchal rigidity	5				
Aphasia	4				All cases showed left-sided clots
Dysarthria	2				
Convulsions	3				1 generalized 2 jacksonian
Clonus (ankle)	2	1		1	
Absent corneal reflexes	1	1			
Astereognosis	1	1			
Diminished hearing	1	1			
Ptosis of eyelid	1	1			
Kernig and Brudzin ki	1				
Divergent strabismus	1				

* McKenzie mentioned questionable disk changes in five cases, one of which he believed appeared within 24 hours.

Diagnosis.—(Table I.) Our case histories were frequently inadequate because many of the patients were unconscious when first found and no reliable story could be obtained. The cause of injury was, therefore, indefinite in the great majority of cases. However, there were only three or four in which the injury could be considered as slight. Most of the known causes were automobile accidents, assaults and falls. It has been impossible to decide from our histories which cases had sustained a general and unlocalized head injury and which had received a local blow to the temporal region.

In 23 of the 44 cases, some mention of the ingestion of alcohol was made. We are well aware of the inaccuracy of such statements as "odor of alcohol," and this finding is merely mentioned as one of the probable factors in confusing the diagnosis.

Those who write of the classical picture of extradural hematoma stress the importance of a "lucid interval." There are many reasons why this may be missing or camouflaged. Alcohol, concomitant brain injury and the amount and type of bleeding all may play a part. In our series, 21 or 40.7 per cent, of the cases showed a fairly definite lucid interval. The most prolonged was an interval of 16 days while the shortest was one and one-half hours. The average was slightly longer than three days, the mean being 48 hours. These latter statistics are very much the same as those of McKenzie.[16] Thus, we may say that in more than half the cases, no history of a "lucid interval" was obtainable.

The age of the patient was definitely mentioned in 38 of our 44 cases. The average was 36 years; the youngest patient being eight and the oldest 74. Table II shows the distribution. It is clear that this condition is rare under ten and uncommon beyond age 50. All but one of our cases were males.

TABLE II

AGE DISTRIBUTION

Age	Number of Cases
1–10	1
10–20	7
20–30	6
30–40	9
40–50	7
50–60	5
60–70	2
70–80	1

The site of the clot was mentioned in 43 cases. Twenty-six were right-sided and 15 left. One was bilateral and one occurred in the left posterior fossa.

The neurologic status of the extremities has been considered important in the diagnosis of extradural hematoma. However, it is our impression that the neurologic findings on any one examination are not as important as the shifting or changing of neurologic signs and cannot, in any event, replace an adequate history. In our series, a definite mention of unilateral weakness of the extremities was made in 25 instances. In 22, it was contralateral to the

clot while in the remaining three the weakness was ipsilateral. Thus, in 12 per cent of the instances in which a unilateral hemiparesis was present it was of no localizing value.

Little mention is made in the literature of facial weakness. In our series we found it mentioned on 13 occasions. It was contralateral to the lesion in 11 instances and ipsilateral in the other two. Both of the latter were specifically described as having the peripheral type of facial weakness, while the former were frequently referred to as of the central type. Thus, it is probably fair to assume that the two instances of ipsilateral facial weakness were due to local damage to the facial canal in the temporal bone.

Babinski's sign was present in 34 cases. It was bilateral in 23, contralateral in ten, and ipsilateral in one. It seems obvious that this sign is of no great localizing value in these cases.

Much has been written about the pupillary changes in extradural hematomata and certain authors lay a great deal of stress on the localizing value of such changes. The so-called Hutchinson's pupil, or a dilated and fixed pupil on the side of the lesion, has been considered important since Hutchinson's[6] original description in 1867. In our series, definite mention of the state of the pupils was found in 37 instances. In 18 (48 per cent) the ipsilateral and in five (13 per cent) the contralateral pupil was dilated. In 13 cases (35 per cent) there was a definite statement that the pupils were normal and equal. In the single instance of a bilateral extradural hematoma, the pupils were first equal after which the right became greater than the left. We may conclude that the unilateral, dilated, fixed pupil (Hutchinson) is by no means as common an occurrence as has been frequently stated. However, when present, it is probably ipsilateral in respect to the clot.

We are well aware of the difficulty in estimating the state of consciousness of an individual, and have already mentioned some of the extraneous factors that may play a part in the picture. Some mention of this symptom and its severity at the time these patients were admitted to the Neurosurgical Service was made in 42 of our 44 cases. In 21 cases (50 per cent) the terms "unconscious," "deeply unconscious," "coma" or "deep stupor" were used. In 15 cases (35 per cent) such terms as "drowsy," "stuporous" or "semistuporous" were used. The remaining six cases were described in the following terms: "Conscious but incoherent"; "amnesia for the accident but conscious"; "alert, oriented but no memory of the accident"; "uncooperative but oriented"; "irrational, resistive and restless"; "semiconscious but responds." In relation to Pringle's[10] statement about prognosis, it is interesting to note that of our 21 cases definitely in coma, 15 (71 per cent) died. In the group of 15 cases described as being drowsy or stuporous on admission, seven cases (46 per cent) died, while in the six cases that were conscious on admission, there was only one fatality.

Pringle lays special stress upon the importance of local hematomata, and bleeding from one ear. He also speaks at some length about percussion of the head over the suspicious area. We have no data on the value of this

procedure. In our series, bleeding from the ear was mentioned on eight occasions. It was ipsilateral in six, contralateral in one, and bilateral in one. Other signs of trauma such as the so-called Battle's sign, scalp hematomata, localized tenderness, ecchymosis of the eyes, and abrasions and lacerations of the scalp, were mentioned in 28 cases. In 23 of these, the signs were ipsilateral, in four contralateral, and in one bilateral. Thus, in over 82 per cent of the cases in which these factors were mentioned they were of definite localizing value.

Cerebrospinal Fluid.—There has been much argument about the value and danger of lumbar puncture in head injuries in general. We believe that the danger is minimal and that the information obtained is not only essential but of a sort that far outweighs any possible risk. Except for the intracranial pressure, however, the cerebrospinal fluid findings are of no value in making the diagnosis of extradural hematoma. They do no more than indicate the amount and type of brain damage. This is important, to be sure, as it is on this complication that the presence and length of the "lucid interval" depends. Even the pressure readings, however, are not entirely reliable as they may be falsified because of toxic dehydration or surgical shock.

Our records mentioned preoperative spinal fluid findings in 38 cases. For purposes of the analysis of the cerebrospinal fluid pressure readings, the first puncture that was made on the Neurosurgical Service is the one we have used for our statistical studies. This was chosen because in several instances, earlier punctures made by the General Surgical and Medical Services failed to give accurate pressure readings. We are aware that this arbitrary use of the first "neurosurgical" puncture may alter slightly the composite picture, for in certain instances a primarily elevated pressure may have been lowered by frequent earlier punctures, while in others an initial low pressure, due to surgical shock or dehydration, may have been raised by treatment. The initial pressure was measured in 36 cases. The average reading was 216 Mm. of water (cerebrospinal fluid). The highest pressure recorded was 500 and the lowest 50 Mm. of water. There were 12 cases (33 per cent) with an initial pressure of 150 or below, while 38 per cent were below 200 Mm. of water. Table III shows the variation in pressure:

TABLE III

PRESSURE RANGE OF CEREBROSPINAL FLUID

Pressure in Mm. Water	No. of Cases	Per Cent
50–100	2	5
100–150	10	28
150–200	2	5
200–300	11	30
300–400	5	13
400–500	6	16

It is obvious that in a third of the cases there was an apparently normal spinal fluid pressure but, without associated data as to the patient's pulse pressure and fluid metabolism, such statistics are valueless.

The character and appearance of the fluid was noted in 37 instances. The following descriptive terms were used: Clear; xanthochromic; pink; slightly bloody; and bloody. Only rarely were any accurate cell counts made. In 19 cases (51 per cent) the fluid was described as either grossly bloody or bloody. Ten cases (27 per cent) showed a fluid described as pink. In five cases (13 per cent), the fluid was xanthochromic, while in three cases (8 per cent) the fluid was clear. It is thus apparent that in 92 per cent of our patients who suffered from an extradural hematoma the associated brain injury was at least as severe as a contusion.

In attempting to correlate the spinal fluid findings with the mortality, it is possible to draw only general conclusions. This is because of the influence of such factors as surgical shock, dehydration, the degree of brain injury, the length of the preoperative period, the technical skill of the operator, the hospital operating room equipment, *etc.* In general, it can be said that patients with complicating severe brain injuries, early, excessively high cerebrospinal fluid pressures, severe toxic dehydration, and any associated shock or hemorrhage do very badly and have a poor chance of survival. Thirty-six per cent of the fatal cases had a preoperative pressure of over 300 Mm. of water, while in the group of recoveries only 21 per cent had pressures above this level. In considering the appearance of the spinal fluid, it appears that 90 per cent of the fatalities showed either bloody or pink fluid, while only 62 per cent of the recoveries had fluids of this character. This discrepancy is even more marked if we use only those cases in which the fluid was described as grossly bloody. Sixty-six per cent of the fatalities showed gross blood, while this finding was present in only 37 per cent of the recoveries. Thus, the extent of the brain damage as measured by the appearance of the cerebrospinal fluid plays a more important rôle in the prognosis than does the increase in cerebrospinal fluid pressure.

Roentgenographic Findings.—It has been our experience that the suspicion of the presence of a cerebral extradural hematoma is one of the two absolute indications for roentgenologic examination. It should be realized, however, that the so-called standard roentgenograms are always unnecessary and often inadequate. This particular examination is made only for the purpose of demonstrating a fracture line in relation to either the middle meningeal artery or to one of the large venous sinuses. No more than one film will be necessary but this film must show not only the fracture but also the groove of the artery in the bone and the shadows of the suspected sinuses. To enable the roentgenologist to make such a film, the surgeon must indicate the side of the head that is to be taken, accept the responsibility for the necessary transportation and handling of the patient, and be prepared to provide enough assistance to ensure absolute immobility of the patient's head during the single exposure. Under these conditions, he can accept a negative film as being as truly negative as is possible. In such circumstances, he will revise his estimate of the diagnostic significance of his other findings, whereas with a positive film—that is, one in which a fracture can be shown to cross any part of the artery

or the sinuses—the balance will be weighted in favor of a positive diagnosis of clot and therefore of immediate operative interference. Although the demonstration of a fracture has no prognostic significance in these cases, its diagnostic importance cannot be overrated. We have yet to see an extradural hematoma in the absence of a fracture of the bone in the immediate vicinity of the bleeding point—providing an adequate search for the latter has been made.

In the series under consideration, preoperative skull roentgenograms were taken in 18 cases (40 per cent). In three instances, the reports were negative for fracture and in each of these a fracture was found either at operation or at autopsy. Operation or autopsy also confirmed the roentgenographic findings in the remaining 15 cases. Of the 26 cases in which no roentgenograms were taken, 16 showed a definite fracture either at operation or autopsy. In ten, no fracture was demonstrated. Six of these were autopsied and four operated upon. It is our opinion that extradural hematomata probably do not occur in the absence of fracture.

Differential Diagnosis.—Detailed analysis of the cases in this study bears out the statement that one of us (D. M.[17]) has repeatedly stressed in the past: The clinical picture of extradural hemorrhage is extremely variable and cannot be depended upon from a diagnostic point of view. Even with the associated aids of history, roentgenographic and cerebrospinal fluid findings, this diagnosis remains one of the most difficult in the whole field of craniocerebral injuries. The impression of simplicity of diagnosis that one gains from the usual text-book description is very dangerous and misleading. The so-called classical picture is extremely rare.

Since Jacobson's article in 1885, little has been added to the list of conditions that may be confused with extradural hematoma. They include cerebral lacerations and contusions; localized cerebral edema; subdural hematoma; depressed skull fracture; and intracerebral hematoma (traumatic or spontaneous). Any of the preceding conditions may be accompanied by an extradural hematoma, however, which, when added to the underlying brain damage, tips the scales in favor of a fatal outcome. The problem of separating the former from the latter is frequently made much more difficult by such added complicating factors as intoxication from alcohol or other drugs, surgical shock and toxic dehydration, all of which alter the clinical and cerebrospinal fluid picture. Thus, it is obvious that in many instances the final diagnosis can only be made after a diagnostic exploratory trephination. This can be performed under local anesthesia with but little risk to the patient. It is justified because when treating a desperate situation such as an extradural hematoma and, moreover, one in which the unoperated mortality is 100 per cent, the risk to which a patient without a clot is thus exposed shrinks into insignificance in comparison with the increase in therapeutic efficiency made available to the individual who has been so unfortunate as to be suffering from extradural bleeding.

Treatment.—As soon as the diagnosis of extradural hemorrhage *cannot be disproved*, the only treatment is an immediate emergency operative procedure.

It is important that the surgeon who undertakes to operate upon these cases be well-prepared both as to mechanical equipment and skilled help. He must have available an adequate suction apparatus and a minimal supply of cranial surgical instruments. A reliable method of administering therapy intravenously must be set up at once by a special team who will have this as their sole responsibility. At least two and usually more compatible blood donors must be available as transfusions are always necessary to treat shock and to replace lost blood. They will have to be given through the intravenous set-up. Directness and speed of action are the deciding factors in bringing these cases to a successful conclusion. These preliminary precautions must be taken in every such case. If the patient is in a hospital that lacks adequate operative equipment or personnel, he should be moved at the earliest possible moment to one that has such equipment. The risk of moving even the sickest patient, is minimal when compared with the danger of operating upon an extradural hemorrhage without adequate equipment and help.

The operation can usually be performed with only local anesthesia, but if the patient is resistive and restless a few centimeters of sodium pentothal intravenously will give adequate relaxation. The ideal approach is the usual subtemporal route. This should be modified to meet individual requirements. After the bone has been removed widely enough to give adequate exposure, the clot should be evacuated as completely as possible by suction. The bleeding point must be adequately controlled. This implies technical exactness and certain occlusion of the bleeding vessel. Arteries should be clipped or tied with silk, and venous bleeding points controlled by the careful placing of adequate muscle stamps. Attempts to stop bleeding in these cases with electrocoagulation is dangerous and will usually lead to recurrence of the clot. If the artery has torn close to its entrance to the skull, the external carotid artery should be tied in the neck if there is any doubt about recurrence of the bleeding because of a slipped ligature. Having safely checked the bleeding, the dura should be opened widely in order to give an adequate decompression. We consider this latter procedure important as a protection against the untoward effects of the usual postoperative edema. The wound should be drained with one or two rubber drains led out through the bottom of the incision. These are adjusted according to the size of the cavity that remains after the clot has been removed. One may be placed inside the dura under the temporal lobe and one in the extradural cavity. They provide drainage for whatever oozing there may be after closure and should be removed in 24 hours. On occasion, quite large amounts of bone may have to be removed to adequately control bleeding, especially when it is coming from a lateral sinus. We feel that the absence of this bone is of no importance in the light of saving the patient's life, and especially so if it is removed in an area in which the temporal muscle can be employed as a covering for the opening.

The postoperative care of the patient is extremely important. He must be followed very closely and the so-called malignant postoperative edema combated with adequate dehydration and lumbar punctures. Coma may persist

for several days postoperatively. Every effort must be made to prevent pulmonary congestion and resulting pneumonia. Adequate fluids must be administered. Not uncommon complications of extradural clots are certain midbrain signs which are probably due to the gross shifting of the entire intracranial contents. Perhaps the most dreaded of these is hyperthermia. In the treatment of this condition we have used frequent tepid sponges with remarkable success. Tepid sponges seem to cause dilatation of the peripheral vessels and facilitate heat loss more readily than the alcohol and ice water sponges previously employed. Removal of all covers, playing an electric fan on the patient while still wet, and cold water or shaved ice enemata have also helped. Erickson[18] has recently given an excellent summary of the possible pathogenesis and treatment of so-called neurogenic hyperthermia. He stresses the importance of recognizing the condition in its inception by the accompanying tachycardia and hyperpnea and instituting treatment early. In the extradural cases, however, the usual first sign is the excessive and steady rise in temperature.

Mortality and Complications.—The mortality from extradural hematomata has always been very high except for the unbelievably good results of J. Hill[1] in 1772. The figure usually quoted to-day is around 50 per cent. This comes from clinics in which there are well-equipped neurosurgical units. There can be no question but that the general mortality is much higher. Our 44 extradural hematomata show a total or case mortality of 59 per cent, and an operative mortality of 52 per cent.

Thirty-eight (86 per cent) were operated upon. The six unoperated cases all died. Twenty of the operated cases died. Table IV gives the mortality statistics for the years covered in this study.

TABLE IV

MORTALITY

Year	Total Mortality	No. of Cases	No. Operated
1932	66	3	3
1933	50	4	3
1934	66	6	6
1935	66	6	5
1936	75	4	3
1937	37	8	8
1938	60	5	4
1939	62	8	6

In the six cases that died without being operated upon, the mortality was 100 per cent. One died before operation because of uncontrollable hemorrhage from some unknown source in the nasopharynx. We consider this an unavoidable fatality. Two cases died almost immediately after admission to the accident floor and before even the barest preparations could be made for operation. The remaining three cases died because of the delay in operating. In one, difficulty was encountered in obtaining operative permission. Of the other two, one was kept on the General Surgical Service for 36 hours before the condition was recognized and neurosurgical consultation requested, and the other died on the Neurosurgical Service with the incorrect diagnosis of

lacerated brain and dehydration. In one-half of these six cases, the diagnosis of extradural hematoma was made before death, while in the other half either a diagnosis of lacerated brain or subdural hematoma was made. At least half of them could have been saved by earlier diagnosis and the more rapid institution of therapy.

In analyzing the 20 cases that died in spite of surgical intervention, we may immediately dismiss one that died several weeks after the operation from unrelated lobar pneumonia. The other 19 cases have been divided into two groups: In the first group, there were six deaths that could be ascribed to technical errors; and in the second, 13 that were caused either by the brain damage which accompanied the clot or by other complications which caused or contributed to the fatal outcome. Among these complications were pneumonia, myocardial failure and uremia.

The six fatalities in the first group follow: In one case which was explored through frontal bur-holes, the clot was not found. In a second instance, the patient had a compound fracture as well as an extradural hemorrhage, neither of which was diagnosed. Both of these cases were seen in 1932–1933, before we had developed our present standard of technic and therapy. The third case was one in which the diagnosis was made and bilateral exploration undertaken, but the exposure was inadequate and failed to demonstrate a large posteriorly-located clot. There were, also, two cases which developed postoperative meningeal infection. The final case in this group was one in which a recurrent extradural hemorrhage developed. Failure to recognize this complication early enough resulted in a fatality. It is important to be on the watch for recurrent extradural hematomata; on one occasion we had removed such a recurrence with ultimate recovery of the patient. The possibility of recurrent bleeding in extradurals has recently been emphasized by Ellis.[20]

The records of the remaining 13 fatal cases demonstrate that death was traceable to the concomitant brain damage (contusion, laceration, and intracortical clot), which was so severe as to make recovery improbable, or to some extraneous complication which of itself prevented recovery.

An analysis of these mortality statistics warrants the following conclusions: There will probably be about 25 per cent of the cases of extradural hematoma in which either the severity of the accompanying brain damage or the general physical condition of the patient will prevent recovery, even with the most timely and ideal treatment of the clot. In another 15 per cent, recovery would be probable if all possible advantages were taken of our present diagnostic and therapeutic methods. Thus, providing ideally proper care had been given, there would have been a reduction of our case mortality from 59 to 44 per cent.

SUMMARY AND CONCLUSIONS

A series of 44 cases of extradural hemorrhage, seen and treated on the Neurosurgical Service of the Boston City Hospital, between the years 1932–1939, has been reviewed.

A statistical study of the clinical, cerebrospinal fluid, and roentgenologic findings has been presented.

A standardized method of treatment has been outlined.

We believe that the so-called classical picture of extradural hematoma is so rare that it is the exception rather than the rule, in practice. This fact must be more widely recognized if the mortality from this condition is to be reduced.

In the past, the possibility of venous bleeding as a source of extradural clots has been overlooked. Sufficient evidence has now been collected to demonstrate its actuality and to prove its importance.

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EXTRADURAL VENOUS HEMORRHAGE

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THE RARE OCCURRENCE of extradural venous hemorrhage in comparison with subdural venous hematoma or extradural arterial hemorrhage made us feel that attention should be attracted again to its symptoms and signs.

In patients having extradural hemorrhage, either arterial or venous, a lucid interval of hours to days usually intervenes between the time of the accident and the onset of symptoms of increased intracranial pressure. The accumulation of extradural venous blood is naturally slower than if the blood were arterial, but as Verbruggen[1] stated, it is the blow to the head at the time of the accident that separates the dura from the bone and bleeding occurs into the space thus formed. He felt that it is the shaking of the dura from the bone and its adherence to the overlying skull in the face of an advancing clot that are the principal factors in the length of the lucid interval. The resultant compression of the brain beneath the extravasated blood usually produces contralateral weakness, altered reflexes, jacksonian seizures and other localizing signs. In many instances the pupil is dilated on the side of the lesion. Restlessness followed by drowsiness and other signs of increased intracranial pressure develop, and surgical intervention becomes a life-saving measure.

Postoperative extradural venous hemorrhage is still a serious complication following craniotomy, and if not recognized may be the important factor in producing a fatality after an otherwise satisfactory operation. Dandy[2] and Poppen[3] have recently emphasized certain operative steps in an attempt to prevent such hematomata, but we occasionally are forced to elevate bone flaps and remove extradural venous clots in spite of employing all precautions. Such postoperative hematomata need be only 1 cm. in thickness, and localized beneath the bone flap, to change a conscious, alert patient to a stuporous, unresponsive one.

Diagnosis.—The deep red color of venous hemorrhage found on trephination is the only accurate method of differentiating a tear of the smaller branches of the middle meningeal artery from a tear in the emissary and meningeal veins. Even then, it may be difficult to decide unless one utilizes the maneuver of Verbruggen in which, when an assistant compresses the common carotid artery on the affected side, arterial bleeding stops and venous bleeding increases. This venous bleeding increases because the internal jugular vein is compressed along with the carotid artery.

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Although extradural venous hemorrhage does occur without linear fracture, suspicion of venous hemorrhage is given roentgenographically if a depressed fracture of the skull is near a dural sinus. The location of linear fractures by overlying edema of the scalp, and by roentgenograms, may give a clue as to whether the middle meningeal artery or the veins entering the lateral or longitudinal sinuses have been torn.

Extradural venous hemorrhage is difficult to differentiate from extradural arterial hemorrhage and acute subdural venous hemorrhage.

Carter[4] stated that when one can elicit a history of a blow on the head: a primary loss of consciousness due to concussion, a clear interval with regaining consciousness, and a second loss of consciousness, the burden of proof is on him to rule out an extradural hemorrhage, and such a history is sufficient to warrant a cranial exploration. He related the case of a two-year-old girl with a linear fracture in the parietal region who showed very little in the way of signs following a fall out of a window, until 48 hours later when she became unconscious and showed weakness of one side. A large extradural hemorrhage was found, with bleeding from several veins near the midline of the skull.

McKenzie[5] emphasized the lucid interval as the most valuable diagnostic sign in extradural arterial bleeding, but a prolonged lucid interval of even one or more days was not infrequent and might confuse the diagnosis. Such cases may become critically ill very rapidly. He found that a dilated pupil, roentgenographic evidence of fracture, and paresis were valuable diagnostic and lateralizing signs. The initial clinical evidence of injury to the brain was insignificant in practically all his cases. Failure to operate and delay in operation caused death in six of his 20 cases of extradural hemorrhage, so that he advocated bilateral exploratory bur-holes if there was a reasonable suspicion of the diagnosis, believing it was better to have a negative exploration than an operation carried out too late or not at all.

Verbruggen felt that the typical and characteristic syndrome of extradural hemorrhage was recognized readily and did not present a special problem, but there was a large group of cases in which the diagnosis was extremely difficult because the extradural clot developed while the patient was unconscious from severe cerebral injury or from alcoholism. He emphasized the facts that the lucid interval may be absent and that the test for hemiplegia even in the comatose patient could be made by firm supra-orbital pressure. He felt attention had been too closely focused on the middle meningeal artery, since a number of his cases had extradural venous hemorrhage from tears in the meningeal veins. He also reviewed the important work of the anatomist Wood Jones,[6] who showed that the grooves on the inner surface of the skull, usually considered as due to the pulsating middle meningeal artery, were actually produced by the veins or sinuses which accompany the artery. Verbruggen believed that the best indications for operative interference in a patient unconscious from cerebral damage were deepening coma and the insidious onset of hemiplegia. When in doubt a bur-hole was not dangerous to the patient and determined the presence or absence of an operable condition.

Ricard[7] reported a case and gave an extended discussion of traumatic rupture of the lateral sinus producing extradural hemorrhage.

Voris'[8] case simulated a middle meningeal hemorrhage, but was due to a tear in the lateral sinus.

Kennedy and Wortis[9] reported 72 cases of acute subdural hematoma and 17 cases of acute epidural hemorrhage operated upon within three weeks of the accident. They noted that acute subdural hematoma occurred at all ages, whereas epidural hemorrhage was more apt to occur in young adult and middle-aged individuals. The epidural hemorrhage usually occurred on the side which received the trauma, whereas an acute subdural hematoma was much more likely to be in the nature of a contrecoup phenomenon. Epidural hemorrhages were almost invariably associated with an overlying fracture line crossing the middle meningeal groove or one of the cranial venous sinuses. The lucid interval in cases of epidural hemorrhage was usually of shorter duration. If paresis resulted in cases of epidural hemorrhage it was practically always contralateral. In cases of acute subdural hematoma a resultant paresis was just as apt to be ipsolateral.

Treatment.—The only treatment for extradural hemorrhage, whether of venous or arterial origin, is prompt surgical intervention. When in doubt as to the proper course to follow after an injury to the head and when the condition of the patient remains precarious or does not improve, we have found that the bilateral exploratory bur-holes, made under local anesthesia, have never done harm and generally have been of valuable diagnostic aid in differentiating between increased intracranial pressure from trauma and edema of the brain or from subdural and extradural hemorrhage.

Extradural venous bleeding has been controlled by gauze packs, by enlarging the bur-holes, or by turning down a bone flap to expose the bleeding points and control the hemorrhage with pieces of muscle.

The two cases herewith presented will emphasize some of the difficulties encountered in treating extradural venous hemorrhage.

CASE REPORTS

Case 1.—(E. J. M.). D. L., male, age 21, on the evening of March 24, 1929, fell while trying to board a moving street car and struck his head on the pavement. He was unconscious for approximately 15 minutes. There was no bleeding from the ears, nose or mouth and no laceration of the scalp. Upon regaining consciousness he went home but that same evening he entered the Mission Emergency Hospital, complaining of headache and nausea. Examination after transference to the San Francisco Hospital showed him conscious, rational and cooperative, with a pulse of 68, respirations 20, and blood pressure 136/64. There were no localizing signs or evidence of increased intracranial pressure. The following day he was rather stuporous with a slower pulse and respiration. On the morning of March 26, 1929, the patient was stuporous, the right pupil was dilated, the pulse 60, and respirations 16. A left facial weakness and fairly marked paresis of the left arm and leg had developed. *Clinical Diagnosis:* Extradural hemorrhage on the right side. Operation undertaken at once.

Operation.—A right subtemporal decompression was planned. On trephining the bone, black clotted blood bulged through the opening. About 20 cc. of clotted blood was removed when the opening was enlarged. This was followed immediately by rather severe

bleeding which filled the cavity. The bleeding seemed to come from the transverse and longitudinal sinuses. The middle meningeal artery was tied but this did not decrease the hemorrhage to any appreciable extent. Gauze packing was inserted in the direction of the sinuses which stopped the bleeding. The patient regained consciousness as soon as the extradural hemorrhage was removed. The wound was closed and the patient was returned to his bed in good condition. The weakness of the left face, arm and leg was hardly noticeable but the right pupil was still dilated.

Subsequent Course.—On March 27, 1929, the pupils were equal, there were no localizing signs, and the patient was conscious, rational and quite cooperative. Approximately 18 hours postoperatively the packing was removed. Within a very short time he became extremely restless, then stuporous. The right pupil was dilated, pulse 58, respirations 18, and blood pressure 186/90.

The patient was again taken to the operating room. When the wound was reopened, a large amount of extradural blood was removed and a thin strip of muscle taken from the thigh was placed along the longitudinal and transverse sinuses. Within a very short period of time bleeding stopped and the wound was closed. The following day the patient was greatly improved. He was clear mentally, his pupils were equal, and the blood pressure was 142/82. The only positive sign was a slight right facial weakness, but within a week no positive neurologic findings remained.

Case 2.—(F. L. R.). E. G., male, age 38, referred by Dr. D. Carson, was struck and knocked down about noon, February 17, 1940. He picked himself up and was taken at once to a hospital, where roentgenograms revealed a fractured left ulna. While this examination was being made, two hours after the injury, his speech became incoherent and a roentgenogram of the skull was made which revealed a linear fracture in the left occipital bone extending from behind the mastoid to the vertex. He became stuporous that afternoon and a lumbar puncture revealed slightly blood-tinged fluid under increased pressure. He remained quite drowsy for the next two days although he could be aroused sufficiently to take small amounts of food. The pupils had been dilated with atropine the day of the accident. During this time the neurologic examination was normal.

He was transferred to Stanford Hospital the evening of the third day, at which time he was stuporous. The pupils were still dilated from atropine and the eyegrounds were not remarkable. The reflexes were equal. Dorsal flexion of the left big toe was obtained on plantar stimulation. Sensory examination could not be made because of the coma. Examination of the roentgenograms taken the day of the injury showed the calcified pineal body displaced a little to the right. New roentgenograms, examined immediately, revealed the pineal body displaced 0.5 cm. to the right and downward. *Clinical Diagnosis:* Probable subdural hematoma. Immediate operation was undertaken.

Operation.—February 20, 1940: Under local anesthesia, bilateral bur-holes were made in the parietal region. A tense brain was seen on the right side but on the left side, as soon as the bone was perforated, old, dark blood escaped, estimated to be four to five ounces. The trephine opening was enlarged, which increased the venous bleeding until sinuses between bone and dura were coagulated. Another bur-hole in the left temporal region revealed no blood. With the release of the extradural blood the patient regained consciousness. Intravenous normal saline, 2,300 cc., was administered slowly to keep the brain and dura tight against the bone. At the end of the operation a transfusion was given.

Subsequent Course.—The patient did well that night but was drowsy the next morning, and did not cooperate well during the neurologic examination.

Two days after operation, on February 22, 1940, he was again stuporous. On reopening the left temporal bur-hole no blood was found. A new trephine opening was made over the left occipital region and dark clotted blood was found to have separated the dura 2 cm. from the skull. Another bur-hole further forward and near the vertex exposed the edge of the clot. A bone flap was then turned down over the left parieto-

occipital region and about four ounces of clotted blood removed. The dura was found separated back to the tentorium and across the vertex, with a number of vigorously bleeding points from the longitudinal sinus. Temporary dry gauze packing decreased the bleeding sufficiently to permit waxing the inner table and controlling the bleeding from the longitudinal sinus with strips of muscle, obtained from the exposed temporal muscle. In the meantime intravenous normal saline had been started and within an hour the depressed brain and dura had expanded to touch the skull. The patient became conscious and started taking fluids. Another blood transfusion was given at the end of this operation.

The next day he was drowsy, but on February 24, 1940, he was alert and cooperated during examination, which revealed a right-sided anesthesia and a right homonymous hemianopsia. He was hesitant in finding proper words. March 3, two weeks after the injury, the positive neurologic findings were no longer present. He returned home on March 13, and resumed work June 1, 1940.

CONCLUSIONS

Extradural hemorrhage requires prompt diagnosis and surgical treatment.

Extradural venous hemorrhage was considered of rare occurrence.

A considerable number of extradural hemorrhages, believed to arise from a torn middle meningeal artery, were caused by torn meningeal veins or sinuses.

A lucid interval followed by stupor, dilated pupil, roentgenographic evidence of fracture, and paresis, are valuable diagnostic signs of extradural hemorrhage.

The only accurate method of differentiating between extradural arterial and extradural venous hemorrhage is the color and source of the blood found on trephination.

Occasionally, exploratory bur-holes, made under local anesthesia, are necessary to differentiate between increased intracranial pressure from trauma and edema of the brain on the one hand, and from subdural and extradural hemorrhage on the other hand.

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THE SURGICAL TREATMENT, BY DRAINAGE, OF SUBACUTE AND CHRONIC PUTRID ABSCESS OF THE LUNG*

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THE VOLUMINOUS literature concerned with chronic abscess of the lung indicates the attention which has been paid to this condition. Unfortunately, it also discloses a general lack of agreement on a plan of management and an absence of any uniformity of view in regard to treatment. Indeed, the reported poor results of operation are so nearly identical with the reported poor results of nonoperative treatment that justifiable confusion exists at the present time as to choice of procedure. The purpose of this communication is to set forth certain principles in the selection of operative cases and in operative treatment, in the hope that some aspects of the surgical management of the disease may be clarified.

It is our opinion that the difficult problem of chronic abscess of the lung should no longer exist. We have maintained that abscess of the lung, in its acute stage, is a surgical disease, that it should not be permitted to pass into the chronic stage, and have demonstrated that the results of operation in the acute stage, both as to life and function, are excellent. For purposes of comparison with the results of operation for subacute and chronic abscess, our results of operation for acute abscess, which have been discussed elsewhere,[3 to 9] should be mentioned. To date, 104 patients have been operated upon for acute putrid abscess, with four deaths. The patients who recovered are well, and the wounds are healed in almost all cases, with the exception of four recently operated upon. All of the latter are progressing satisfactorily. However, there is as yet no general acceptance of our views on early operation, and we assume that chronic pulmonary abscess will remain a problem for years to come.

Definition.—The term "chronic putrid abscess of the lung," as employed in this paper, should be clearly defined in order to avoid confusion referable to the inclusion of heterogeneous cases. Consideration, therefore, will not be given to nonputrid chronic pulmonary abscess, pulmonary abscess due to extension of infection from neighboring regions, pulmonary abscess due to aspirated foreign body, and pulmonary abscess secondary to wounds, carcinoma, or tuberculosis. Putrid pulmonary suppuration secondary to bronchiectasis is also excluded. Thus, there remains for discussion a lesion which

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may be termed "primary" putrid abscess of the lung, ascribed by us to known or assumed aspiration.

Shall subacute and chronic abscess be defined from the point of view of time or of pathology? In our presentations of the subject of acute putrid abscess of the lung, we pointed out that the majority of lesions remained single and uncomplicated during the first six weeks of the disease. Accordingly, we defined acute abscess rather empirically as one of less than six weeks' duration. Occasionally spillover bronchopneumonia and multiloculation occurred during this period. However, multiloculation, spillover bronchopneumonia, gangrenous extension, the formation of additional abscesses, and beginning surrounding fibrosis occurred more frequently in the second stage of the disease. These features, although not regularly present, nevertheless characterize "subacute abscess of the lung" which we have designated rather arbitrarily as extending over a period of six weeks (from the beginning of the seventh week to the end of the twelfth week). All abscesses of more than 12 weeks' duration are termed "chronic." Chronic putrid abscess of the lung presents the features of subacute abscess with the addition of well established surrounding pulmonary fibrosis and bronchiectasis. It will be shown in a succeeding paragraph, however, that the pathology of chronic pulmonary abscess may present considerable variation.

Comments on Some Current Methods of Treatment.—So-called conservative methods of treatment, as employed in cases of acute abscess of the lung, have been applied to subacute and chronic abscess. As a result, there is to be found, even in recent literature, the recommendation of drug treatment, fresh air and rest, postural drainage, bronchoscopic treatment, pneumothorax, and thoracoplasty. We are of the opinion that such methods offer little likelihood of cure of pulmonary abscess. Assuming their possible curative effect in the acute stage, little more than some measure of relief can be anticipated from their employment in subacute and more particularly in chronic abscess. In addition, there is grave risk in the application of pneumothorax or thoracoplasty.

The only method of treatment to be discussed in this paper is the time-honored one of surgical drainage. That it is far from ideal is very generally recognized. The reported operative mortality is high, ranging from 25 to 50 per cent in different statistics. Furthermore, reported results in patients who survive are by no means uniformly good. The analysis of 47 operative cases by Cutler and Gross[1] in 1936, is of interest in this connection. Sixteen patients were cured, six improved, three unimproved, and 22 died. In fact, these authors reported better results and lower mortality (29 per cent) in 38 patients who received medical treatment. Thus, the question to be answered is whether surgical drainage, which is in general use at the present time in the treatment of subacute and chronic abscess, is a justifiable procedure. We believe that the answer lies in the correct selection of cases, to be followed by precise localization of the lesion, and the establishment of drainage by means of an adequate unroofing operation. The question of selection of

suitable cases for drainage will be discussed under the headings of subacute and chronic abscess of the lung.

Subacute Putrid Abscess.—From the viewpoint of pathology, subacute putrid abscess may present features which are common either to acute or chronic pulmonary abscess. Thus, the cases fall essentially into two groups. In the first group the features resemble those of acute abscess because the lesion is essentially localized. Although the cavity frequently is multilocular, there is only a limited tendency for the spread of pulmonary infiltration beyond its original site. In the second group of cases, the lesion resembles chronic abscess insofar as there is a tendency for the pneumonitis to spread by direct extension into adjacent areas of the lung and by the mechanism of spillover into distant areas. In some cases this acute gangrenous extension may be so widespread that death ensues regardless of adequate surgical drainage of the abscess. In other cases of the second group, pneumonic spread occurs more slowly and the lesion insidiously acquires the characteristic features of the chronic stage, namely, induration, fibrosis, multiple cavity formation, and bronchiectasis. These features, when present, are to be noted only in

TABLE I

RESULTS OF DRAINAGE OPERATIONS

A. Localized Lesions

	Totals	Cured	Improved	Dead	Inadequate Follow-Up
Subacute	25	19	0	6	0
Primary chronic	31	23	3	4 (1 suicide)	1
Secondary chronic	7	5	1	0	1
Totals	63	47	4	10	2

B. Diffuse Lesions

Subacute	6	0	0	6	0
Primary chronic	26	5	6	12	3
Secondary chronic	9	0	2	7	0
Totals	41	5	8	25	3

C. Putrid Empyema or Pyopneumothorax

(Differentiation between localized and diffuse pulmonary lesions not possible)

Totals	15	5	2	7	1

the later part of the subacute stage. On the other hand, the localized type of lesion may be encountered throughout the subacute phase.

From the foregoing, it is clear that the selection of cases of subacute abscess suitable for drainage operations should be based upon the differentiation between the localized and diffuse forms of the disease. The methods to be employed in attempts to differentiate between the two types will be discussed separately. They consist of roentgenography and bronchoscopy, and bronchography under special circumstances.

In our series of 31 operative cases of subacute putrid pulmonary abscess, no selection was made in accordance with the known or assumed type of lesion, because the importance of this differentiation was not realized at the time. The lesion found at operation in 25 cases was of the localized type, and in six was of the diffuse type (Table I). Although the most important criterion of operability by drainage should have been a localized cavity with comparatively little surrounding pulmonary infiltration, fibrosis, and bronchiectasis, drainage operations were attempted or performed in six cases which would be classified to-day as unsuitable for this type of operative procedure. These cases were operated upon during a period in which alternative worth while operative procedures had not as yet been developed.

Of our 31 operative cases of subacute putrid pulmonary abscess, 19 were cured and 12 died (Table I). In all 19 cured cases, the lesion was of the localized variety, proving that surgical drainage is an effective form of therapy in this type of case. In six of the 12 cases that ended fatally, the lesion was of the localized type. Death due to pleural infection occurred in four of these cases, because of pleural entry at the time of operation; and in two cases death resulted from spillover gangrenous bronchopneumonia. We believe that the deaths due to pleural infection would probably not occur to-day because of precise methods of localization and our present operative method of dealing with the opened pleura.

Of the six fatal cases with lesions of the diffuse type prior to operation, three died of pleural infection which also might have been avoided. In the remaining three, the fatal termination was due to spillover gangrenous bronchopneumonia, which probably was unavoidable (Table II). In short, in seven of the 12 fatal cases death was due to postoperative pleural infection alone or in combination with some other infective lesion (suppurative pericarditis, mediastinitis, spillover bronchopneumonia).

The remaining five cases, in which death was due solely to spillover gangrenous bronchopneumonia, require special consideration (Table II). Spillover gangrenous bronchopneumonia is not uncommon in subacute abscess of the lung, and is one of the great dangers in that stage of the disease. It may occur spontaneously during this phase or may be precipitated by operation. Further emphasis is placed on the importance of spillover bronchopneumonia by the fact that this complication was a contributing factor to the mortality in several of the cases in which pleural or other infections occurred after operation.

Chronic Abscess.—At the time of operation, approximately 80 per cent of the cases of chronic abscess of the lung in our series were of four to 12 months' duration. The remainder had lasted from two to nine years. As in subacute abscess, the cases were of the localized and diffuse varieties. The localized form was not distinguishable, pathologically, from that noted in subacute abscess. In the diffuse form the lesion extended beyond one bronchopulmonary segment and usually was accompanied by considerable pulmonary infiltration, fibrosis, and bronchiectasis. Not infrequently there were multiple

abscesses or involvement of several lobes and, at times, of the opposite lung. Occasionally the original pulmonary abscess was more or less completely obscured by extensive induration and fibrosis.

TABLE II

CAUSES OF DEATH

	Localized Type	Diffuse Type	Totals
Subacute Cases			
Pleural infection	4	0	4
Pleural infection plus mediastinitis	0	1	1
Pleural infection plus pericarditis	0	1	1
Pleural infection plus spillover gangrenous bronchopneumonia	0	1	1
Spillover gangrenous bronchopneumonia	2	3	5
Totals	6	6	12
Primary Chronic Cases			
Cerebral metastases	1	4	5
Sudden death on table (cerebral embolism?)	1	3	4
Metastatic renal abscess	1	1	2
Hemorrhage	0	1	1
Empyema	0	1	1
Gangrenous spillover bronchopneumonia	0	1	1
Suicide	1	0	1
Operated upon in moribund condition	0	1	1
Totals	4	12	16
Secondary Chronic Cases			
Cerebral metastases	0	3	3
Spillover gangrenous bronchopneumonia	0	2	2
Sepsis plus spillover gangrenous bronchopneumonia	0	1	1
Pericarditis ? (no autopsy)	0	1	1
Totals	0	7	7
Perforated Cases			
Sepsis (one due to phlebitis following intravenous therapy)			2
Cerebral metastasis			1
Pulmonary suppuration			1
Spillover gangrenous bronchopneumonia plus pyopneumothorax (contralateral)			1
Cardiac failure			1
Pericarditis			1
Total			7

It is significant that more than half of the lesions were of the localized type. Thus, a substantial proportion of cases of chronic abscess were suit-

able for drainage operations, according to the view which we hold at the present time.

In contrast to the subacute stage, the operative morbidity and, ultimately, the mortality in the chronic stage was due in part to the fact that the disease had progressed to such an extent that adequate surgical drainage did not suffice. In addition, the operative procedure itself often was the direct cause of a fatal complication. We refer, specifically, to the mortality from cerebral embolization initiated by exploratory aspiration through, or hemorrhage from, infiltrated pulmonary tissue (Table II). Indeed, the danger of immediate or postoperative fatal cerebral embolism exists in any chronic case in which a drainage operation is performed if densely infiltrated pulmonary tissue is traversed in order to enter an abscess cavity. The Trendelenburg position at the time of operation, and the use of the cautery instead of the scalpel, may reduce the shockingly high incidence of cerebral complications, but offer no assurance against its occurrence in the presence of densely infiltrated lung. In contrast to the high mortality of operation upon diffuse lesions, the low mortality of operation upon localized abscesses warrants emphasis (Table I).

A contrast is also to be noted between the results which were obtained in surviving patients of both groups, for there was a much higher proportion of cures after operation in cases of localized abscess (Table I). However, improvement rather than cure (absence of symptoms, disappearance of pulmonary infiltration, healed wound) may be the outcome in any case after operation for chronic abscess. Thus, patients who survive operation may be left with a bronchocutaneous fistula or "lattice lung," or with symptoms referable to chronic pulmonary fibrosis or bronchiectasis.

Differentiation between Localized and Diffuse Lesions.—Since the differentiation between the two varieties of subacute and chronic abscess is of clinical importance, the methods to be employed for the purpose require at least brief discussion. Typical localized abscess is recognized readily on roentgenography. When the roentgenogram is not characteristic, however, particularly if the cavity is more or less completely obscured by pulmonary infiltration or pleural reaction, the diagnosis of a localized lesion may be difficult. Under these circumstances, bronchoscopic examination (and occasionally bronchography) often will establish the diagnosis of a localized abscess by revealing the escape of pus from a single bronchopulmonary segment. However, the discharge of pus from two adjacent segments does not exclude the existence of a localized cavity. For example, the abscess may bridge an interlobar fissure. On the other hand, the discharge of pus from unrelated segments indicates a diffuse lesion or a multiplicity of lesions.

The diagnosis of a diffuse lesion can usually be made by a study of the roentgenogram combined with bronchoscopic examination. It is important to remember that the interpretation of roentgenograms may be faulty, especially in chronic cases, because of shadows due to pleural involvement. If doubt exists as to whether or not a lesion is of the localized type, one should proceed on the assumption of a localized lesion.

In a few instances good results followed drainage of diffuse lesions (Table I). Emphasis, however, should be placed on the fact that these patients required multiple operations, which were often hazardous, that serious complications occurred, and that the postoperative course sometimes was unsatisfactory for long periods. Therefore, the conclusion is justified that drainage operations should not be employed in cases of abscess of the lung of the diffuse type. Whether more radical surgical procedures such as lobectomy or pneumonectomy meet the requirement of good results with low mortality remains to be seen.

Secondary Cases of Abscess of the Lung.—This comprises a series of 16 cases first operated upon elsewhere for chronic pulmonary abscess or a complicating putrid pleural infection (Table I). They presented special problems in diagnosis, localization, and operative management, and, therefore, are discussed separately. In some cases, pneumonotomy for drainage of the pulmonary abscess was performed several times before admission. The most significant fact is that in nine of the 16 cases of which this group is comprised, the sole operation performed before admission was drainage for empyema. In most of these instances, drainage was carried out in the acute or subacute stage of the disease with *apparent* cure in several cases. However, in such cases, symptoms of chronic abscess subsequently made their appearance after symptom-free intervals. At the time of admission to the hospital all were definitely chronic. The localization of the abscess may be difficult or even impossible from a roentgenologic study, if the lesion is obscured by thickened pleura. Examination of roentgenograms made in earlier stages of the disease, prior to the first operation, may prove of great value in this connection. Localization of the lesion or lesions by bronchoscopy is almost always indicated in these cases, and is of particular value if roentgenographic localization is difficult. The series of cases is too small to permit of statistical analysis, but there were approximately the same number of cases in the localized and diffuse groups. It is significant that all cases of localized abscess recovered after operation, whereas all cases of diffuse abscess, with one exception, died after operation.

Putrid Pyopneumothorax or Empyema Unrelated to Surgical Intervention.—This may occur at any time during the course of subacute or chronic abscess. In this series it was most common in advanced chronic cases and often was an ominous complication (Table I). A study of our cases reveals the interesting fact that the pleural infection was usually, if not always, derived from an *acute* pulmonary abscess complicating the chronic lesion. Thus, the empyema (or pyopneumothorax) was situated either immediately adjacent to the chronic pulmonary abscess and was derived from a recent gangrenous extension into adjacent parenchyma, or when situated at some point distant to the chronic abscess was derived from spillover infection. This group of cases should be classified separately for two reasons: First, because they may present separate problems; and second, because they are often terminal compli-

cations of chronic pulmonary abscess. The results of their treatment are to be found in Table I.

Technic of the Drainage Operation.—The operation which we have employed in cases of subacute and chronic abscess is similar to that which we have developed and utilized in cases of acute abscess. It is based upon two features of our concept of the pathology of the disease: (1) Superficial situation of the abscess within a pulmonary lobe; and (2) the constant presence of overlying adhesions which are most commonly visceroparietal (costal). The operative procedure consists essentially of a one-stage operation in which the abscess cavity is entered through the zone of visceroparietal adhesions, unroofed, and packed with gauze. The details of the procedure, which have been published elsewhere, will not be repeated. Certain features, however, appear worthy of reiteration. The Trendelenburg position is favored because it may reduce the incidence of cerebral embolism. The operative approach is made at the site at which the pulmonary lesion is in contact with the thoracic parietes. Rib resection is of limited extent in order to avoid unnecessary exposure of the pleura beyond the zone of limiting adhesions. As a rule, only a portion of one rib is removed, but occasionally a segment of a second rib is removed in order to facilitate adequate unroofing of large lesions. Since the cavity is usually to be found superficially within the pulmonary substance, if the site of approach is correct, aspiration in order to locate the abscess should be performed under the guidance of the roentgenogram. The danger inherent in aspiration of indurated lung has already been stressed. Therefore, the direction and depth of aspiration should be considered carefully each time it is performed. When the cavity is located, it is entered with a specially designed grooved director and double-edged scissors, and evacuated by suction. The interior then is inspected under direct illumination with a sterile light, and a search made for recesses and communicating loculations. The roof of the cavity and of any adjacent superficially situated loculations is excised. Communicating loculations in the depths are dealt with by providing a free opening between them and the main cavity. Bleeding, which may occur during this step, should be controlled promptly by temporary pressure in an attempt to avoid cerebral embolism. After all recesses and loculations have been cared for, the abscess cavity and its recesses are packed with gauze.

In the occasional case in which the lesion does not face the thoracic parietes, but faces one of the interlobar fissures, the mediastinum, or diaphragm, adhesions between the lung and the parietal pleura are usually absent or insignificant. Under such circumstances, originally we performed the traditional two-stage operation, but finding the results unsatisfactory, the procedure was modified. At present, we suture the lung into the operative wound and proceed with the evacuation of the abscess in one stage.

In cases in which the free pleura was accidentally entered during the course of operation, we formerly attempted to shut off the pleural opening by packing with gauze. This was followed by a high incidence of pleural infection, with high mortality (Table II). Subsequently, all accidental pleural

openings were closed by suturing the lung to the margins of the pleural defect and the overlying soft parts. Since employing this method, there have been no pleural infections.

In a few of the subacute and in many of the chronic cases, multiple drainage operations were performed. The lesions which were opened secondarily either were present at the time of the original operation or else developed subsequently. Depending upon their sites, some were opened through the original operative wound, while others were opened through new incisions. The large number of secondary operations necessary in subacute and chronic cases as compared with the small number in acute cases, is the best commentary on the increasing incidence of multilocular and multiple lesions as time progresses.

In previous communications we stressed the fact that in cases of acute abscess practically all bronchial fistulae closed spontaneously, and that one of the chief problems in postoperative management was that of keeping the fistula open for a sufficiently long period. This, as a rule, is not a problem after operation in cases of chronic abscess. In the latter, the pulmonary cavity is smooth-walled, and, while the cavity may become smaller, it often presents the mouths of one or more discharging bronchial fistulae. In such cases it is our custom to keep the external communication open with a tube until all traces of anaerobic infection have disappeared. In chronic cases of long standing this may require many months. Experience has demonstrated that in cases of doubt, it always is wiser to leave the tube in place rather than to risk premature closure of the wound and recrudescence of infection. During the months that the tube is in place, the superficial portion of the fistulous tract not infrequently becomes lined by skin so that a bronchopulmonocutaneous fistula results. When this occurs, the cavity and the fistulous tract may require some type of plastic closure. This we have accomplished, most commonly, by the use of a free fat transplant which has yielded satisfactory results in a high percentage of cases.

SUMMARY.—Subacute abscess is defined arbitrarily as one of seven to 12 weeks' duration. A chronic abscess is defined as one of more than 12 weeks' duration.

Subacute and chronic abscess may be of either the "localized" or "diffuse" type. The localized type is characterized by a mono- or multilocular cavity with limited surrounding pulmonary infiltration. The diffuse type is characterized by multilocular or multiple cavities with more or less extensive surrounding pulmonary infiltration, induration, fibrosis, and bronchiectasis.

The great majority of primary (previously unoperated) cases of subacute abscess were of the localized variety. Approximately half of the chronic abscesses, whether primary or secondary, were of the localized type.

Operations in all cases consisted of drainage or attempts at drainage. Cure resulted after operation upon the localized form of subacute pulmonary abscess in all patients who survived. Operation on the diffuse form of subacute pulmonary abscess was fatal in all cases. The chief causes of mortality after

operation for subacute abscess were pleural infection, which is avoidable, and spillover gangrenous bronchopneumonia, which is probably unavoidable.

The results of operation upon the localized form of chronic abscess, whether primary or secondary, were good. On the other hand, the results of operation upon the diffuse form of chronic abscess, whether primary or secondary, were bad, and the mortality was very high.

The differentiation between the localized and the diffuse form of subacute and chronic pulmonary abscess is based upon roentgenography and bronchoscopy, and bronchography in selected cases.

Subacute and chronic putrid pulmonary abscess complicated by putrid pleural infection presents special features and comprises a separate problem.

CONCLUSIONS

Subacute and chronic pulmonary abscess occurs in localized and diffuse form. The great preponderance of cases of subacute putrid pulmonary abscess, and a surprisingly large proportion of chronic cases, are of the localized type and are amenable to cure by surgical drainage. The results are usually good. The mortality should be low, since the most common cause of death after operation—pleural infection—is avoidable with precise preoperative localization of the lesion and adequate surgical technic.

Cases of subacute and chronic putrid pulmonary abscess of the diffuse type rarely are amenable to cure by surgical drainage. The results of drainage operations usually are poor because the disease is widespread, and the mortality is high.

The differentiation between the localized and diffuse forms of subacute and chronic abscess is of basic importance in selecting cases for drainage procedures.

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DISCUSSION.—DR. ADRIAN VAN S. LAMBERT (New York) objected to the term putrid in lieu of acute abscess, and felt that patients with an acute abscess should be treated as surgical cases from the beginning, with the surgeon calling in a medical consultant when required. Doctor Lambert also emphasized that no region in the body, except possibly the skull, requires such accurate localization of the lesion as the chest, and upon such localization often depends the success or failure of the case in the operating room.

Doctor Lambert also said that the period of time adhered to by many for conservative treatment of lung abscesses had often been too long—to the detriment of the patient. The inclination at Bellevue Hospital is to watch the patient with an acute abscess for a certain period of time, and not to operate without careful study and consideration, but three months is usually too long, and perhaps even six weeks is also. Three factors have brought about the prevailing point of view at Bellevue, and these are: First, in the presence of the Friedländer bacillus the patient is not operated upon as though the case were one of acute abscess. Secondly, at Bellevue, a number of water-front cases are brought in after submersion, during which the patient has drunk or otherwise inhaled foul sewage from the East River, and within three or four days a fluid level is present. To operate upon such a person has proved unwise. In the few cases that have been operated upon there have not been any adhesions. Doctor Lambert said that in his experience not all acute abscesses are adherent to the pleura, and even a roentgenologic examination will not provide absolute criteria in determining the presence or absence of adhesions unless there is air in the pleural cavity. A third reason for not immediately plunging into an abscess, is because a number of cases come in every year with evidence of acute suppurative disease, who, upon study, prove to have had a previous bronchiectasis for many years, undiagnosed, unnoticed, or forgotten. Attempting to effect simple drainage of an acute lesion in such a patient is one of the reasons operations in chronic abscess cases have been so unsatisfactory. Doctor Lambert said he preferred to wait and perform a lobectomy and excision in these cases.

DR. HAROLD NEUHOF (closing) said that the crux of the matter rested upon whether an abscess is or is not putrid. In employing the term putrid for abscess of the lung, he referred to a very definite type of abscess. One could, he supposed, use the term anaerobic, but neither he nor Doctor Touroff felt free to do so until adequate bacteriologic evidence of the anaerobic nature of the infection could be established. In nonputrid abscesses of the lung the result of infection by streptococci, pneumococci, staphylococci, or other aerobic organisms, the lesion is an area of pneumonia in which more or less widespread suppuration exists. Only in occasional instances is there a well-defined, well-circumscribed collection of pus with limited surrounding pulmonary infiltration. The problem presented by these cases, and their general management, is quite different and most of them should not be the subject of surgical consideration. Spontaneous subsidence occurs frequently, and the chief surgical significance of aerobic abscess of the lung perhaps rests on occasional perforation into the pleura with the formation of a pyopneumothorax. Thus, these are not the cases referred to in the paper by himself and Doctor Touroff.

Putrid abscess of the lung begins as a gangrenous bronchopneumonia which, however, breaks down and forms an abscess within a week or ten days. There is usually only a limited surrounding zone of pneumonitis. The abscess is always of substantial proportions and, being segmental in nature and occupying much of a bronchopulmonary segment, must, of necessity, reach to

the periphery. In reaching to the periphery, there must, of necessity, be an overlying pleuritis. This pleuritis leads, invariably, to shutting off pleural adhesions. The latter are visceroparietal in the great majority of instances. Exceptionally, because of the unusual situation of the abscess, adhesions are from lobe to lobe across a fissure, or from lobe to diaphragm, or from lobe to mediastinum. Surgically speaking, it, therefore, becomes a matter of deciding where the adhesions exist, not if they exist. Not only were they present in all the ten cases of acute abscess upon which Doctor Neuhof and Doctor Touroff had operated, but were sufficiently firm and widespread to warrant a one-stage operation in the great majority of instances.

OBLITERATION OF THE PLEURAL SPACE FOLLOWING PNEUMONECTOMY*

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THE CHIEF INDICATION for pneumonectomy up to the present is the presence of cancer of the lung. Recognition of malignant lung tumor first appeared in the writings of Agricola,[1] in the early sixteenth century. Bell is credited with making the first diagnosis of *primary* lung tumor, in 1846. His case is now classified as lung sarcoma.[1] The first successful treatment of bronchogenic carcinoma was not until 1933, when Graham removed the entire left lung for a primary growth in the left main stem bronchus. The patient, a doctor, is asymptomatic and is actively engaged in practice seven and one-half years following the operation.

The early recognition of lung tumor from clinical symptoms, bronchoscopic, roentgenologic and bronchographic examinations has led to frequent exploratory thoracotomy. Operable carcinomata of the lung are thus discovered earlier, and are now quite often successfully removed surgically. The improvement of the surgical technic of pneumonectomy has been largely due to the efforts of such courageous surgeons as Graham, Churchill and Rienhoff, in America, and Edwards and Roberts, in England. Graham's original pneumonectomy was accompanied by an extensive thoracoplasty to obliterate the remaining pleural space. The operation, as it is usually performed to-day, seldom entails the removal of more than one rib. The remaining pleural space is only partially obliterated by paralyzing the diaphragm.

The changes in respiratory physiology, following total ablation of one lung with the anatomic readjustment in the thoracic cage, are not well understood. The dangers of sudden shift of the mediastinum in intrathoracic surgery has been greatly feared in academic circles. This has resulted, in part, from experiences with the original Brauer thoracoplasty for pulmonary tuberculosis. Many of these patients developed a mediastinal flutter due to an unstable chest wall following extensive rib resection. The principal factor, however, has been the lack of understanding of normal cardiorespiratory physiology and its alteration under abnormal conditions such as an open pneumothorax. Graham's physiologic studies, in 1918, demonstrated con-

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clusively the importance of vital capacity (amount of functioning lung tissue) in the presence of an open pleural cavity. Reports from other clinics, as well as experience in this clinic, indicate that compression of the great vessels resulting from marked deviation is the principal danger from mediastinal shift when an adequate amount of lung tissue is functioning. We have had one instance where definite circulatory and respiratory embarrassment did result from spontaneous pressure pneumothorax in a surgically produced extrapleural space. Mediastinal shift was only of secondary importance in this case. Relieving the pressure pneumothorax brought about immediate alleviation of the symptoms. Untoward symptoms of mediastinal shift following lobectomy, pneumonectomy, or thoracoplasty have not been met with in this clinic. That there has been definite shift of the mediastinum is well known both from roentgenographic and autopsy observations.

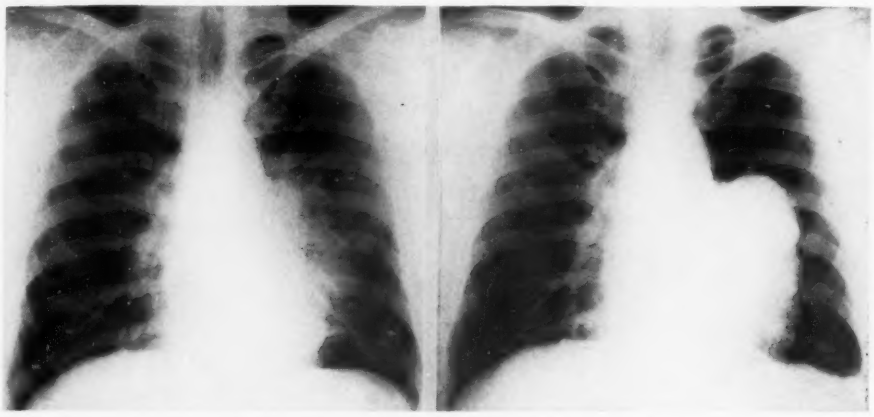
In pneumonectomy for carcinoma of the lung a preoperative pneumothorax is produced to collapse the entire lung on the involved side as much as possible before attempting the operation. This collapse brings about diminished blood flow through the lung, allowing respiratory compensation to occur gradually, and tends to cause thickening of the pleura of the chest wall and of the mediastinum. Bloch[2] has shown that in rabbits the mediastinum does not thicken during pneumothorax. No reported studies, so far, have proven that the human mediastinum does thicken during pneumothorax, but clinical observation at the time of operation, as well as roentgenologic examination, indicates that at least the mediastinal pleura does thicken appreciably if pneumothorax is of long duration. It is doubtful that the short period of immediate preoperative pneumothorax stimulates thickening or fixation of the mediastinum to any demonstrable degree.

Following pneumonectomy there is an accumulation and stasis of a bloody, serous exudate that develops in the pleural cavity and with this a gradual reabsorption of the remaining air. The exudate slowly becomes partially organized and may lead to the formation of fibrous tissue. The parietal pleura becomes markedly thickened. During the process the chest wall on the involved side undergoes shrinkage and the pleural space becomes partially obliterated. The degree to which this process continues varies with the age of the individual and other physical factors which are not well understood. The thickening of the "shell" of fibrous tissue in the ensuing months after operation produces a progressive increase in the contraction of the chest wall. In the case to be reported there was an incased pocket of fluid in this cavity. During the first few weeks following operation this fluid may become infected. If so, the cavity is not likely to become obliterated spontaneously, but will do so following a thoracoplasty, an operation similar to that employed in obliterating a chronic empyema cavity. Rienhoff[3, 4, 5] has found it unnecessary either to drain these cavities or to perform thoracoplasty to collapse them if no infection ensues, and at the present time this is the procedure employed by most surgeons. F. E. Hambrecht[6] used closed drainage of an infected pleural space following pneu-

monectomy for bronchogenic carcinoma in one case, which resulted in complete healing without thoracoplasty.

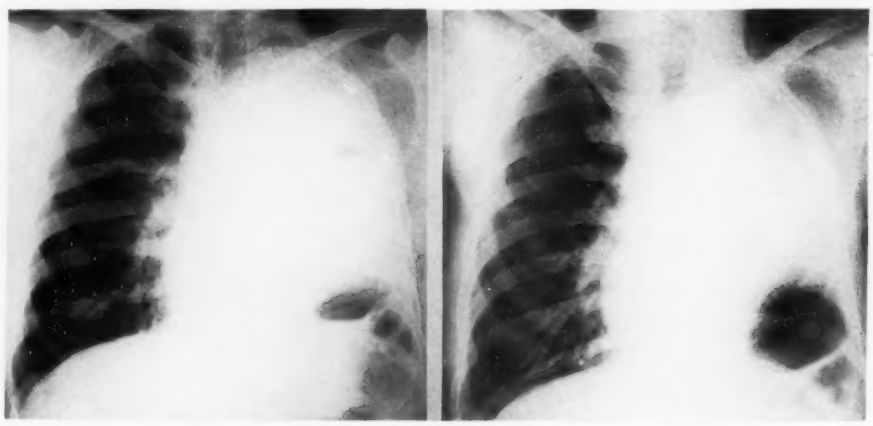
We are reporting a case that died of an unrelated cause nine months following a pneumonectomy for carcinoma of the left lung.

Case Report.—Hosp. No. 191716: H. R., white, male, age 58, was admitted to the University of Chicago Clinics, January 26, 1938, complaining of cough with blood-tinged sputum for one year, night sweats for three months, and a loss of ten pounds in weight.



A B

FIG. 1.—(A) Roentgenogram of chest on admission. Note the opacity extending outward from the left hilum. This is characteristic of pneumonitis and atelectasis resulting from bronchial obstruction.
(B) Roentgenogram of chest following preoperative collapse of the left lung by pneumothorax.



A B

FIG. 2.—(A) Roentenogram of chest 30 days following extirpation of the left lung. The left diaphragm is elevated, and a serosanguineous exudate has almost completely replaced the air in the left pleural space.
(B) Roentgenogram of chest eight months after left pneumonectomy. The left diaphragm is considerably elevated, the interspaces narrowed, and the superior mediastinum deviated toward the left side.

A roentgenogram of the chest showed an opacity, suggestive of atelectasis and pneumonitis of the left hilar region of the lung (Fig. 1 A). Bronchoscopic examination and biopsy proved the diagnosis of squamous cell carcinoma of the left main bronchus. The left lung was collapsed by pneumothorax preceding the operation (Fig. 1 B).

The entire left lung was removed February 21, 1938. The postoperative course was relatively uneventful. He was discharged 42 days after operation. Three months after operation he was given 3,028 r units in divided doses over the mediastinal area. There was a very mild reaction to the irradiation. Fluoroscopic examinations made at that time showed a marked increase in density of the left pleural space.

Throughout the ensuing months he had a mild, persistent cough, occasionally raising a small amount of mucus-like material. He gained three pounds in weight and was otherwise in good health generally.

Bronchoscopy performed four months after operation showed no ulceration, carcinomatous tissue or evidence of opening of the bronchial stump of the resected lung. A roentgenologic examination of the chest showed only an elevated diaphragm and a marked increase in density on that side (Fig. 2 A). In a subsequent roentgenologic examination, eight months after operation, there was an increase in the density of the left side of the thorax. The left diaphragm was higher than at the previous examination (Fig. 2 B). The patient continued in general good health.

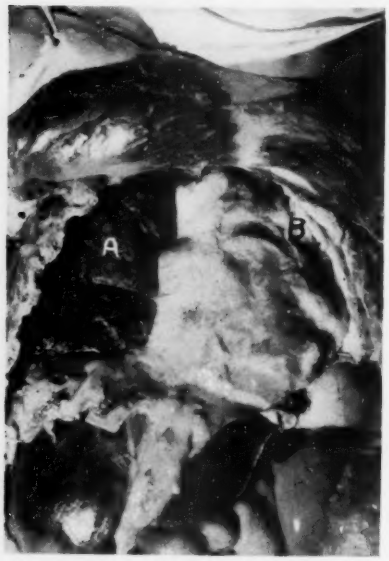


FIG. 3.—Anterior view of the contents of the thorax after removal of the sternum and costal cartilages at autopsy. The right lung (A) appears normal. The contracted left pleural space (B) is obliterated by a mass of fibrous tissue. The pericardial sac was completely obliterated by adhesions.

Nine months after operation he developed acute abdominal pain, followed by nausea, vomiting and fever. He was first examined about 40 hours after the onset. A diagnosis of an acute surgical abdomen was made. At exploratory celiotomy, a gangrenous appendix with general peritonitis was found. He died three days later of the peritonitis.

Autopsy.—Path. No. 4581: Dr. Paul R. Cannon. The entire left chest cavity was lined with a well-organized, dense, fibrous labyrinthine shell which contained a brownish, turbid sterile fluid. This shell varied from 0.5 to 2.0 cm. in thickness. The right lung exhibited no gross evidence of emphysema. The heart was slightly enlarged and adherent to the fibrous tissue shell. The pericardial cavity was completely obliterated. There was moderate collapse of the left pleural cavity by contraction of the chest wall (Fig. 3). The hilar lymph nodes at the stump of the left main bronchus contained carcinoma cells. There was no evidence of distal metastases of the tumor. Microscopically, the wall of the shell showed only fibrous tissue with occasional fibrocytes.

In an effort to produce and explain similar respiratory changes, we have carried out left-sided pneumonectomies upon dogs and have studied their reaction to the diminished lung capacity. In very few instances was there evidence of dyspnea. The hemoglobin and hematocrit readings increased immediately after pneumonectomy in most dogs, but returned to normal in a few weeks. About six weeks after the pneumonectomy we began to effect stenoses of the bronchi of the three most dependent lobes of the remaining right lung. Several of these animals are apparently quite normal, even though only the right upper lobe is functioning. Unlike adult man, the dog's mediastinum is very mobile. Following the resection of an entire

lung with gradual resorption of the remaining air in the pleural space, the mediastinum is slowly deviated toward the chest wall of that side. Very little pleural exudate develops and the parietal pleura exhibits little or no increase in thickness.

The clinical case presented is especially valuable as a specimen for study of the anatomic changes that occur in the chest following removal of one entire lung. This patient had a well-organized, unnecessary fibrous cone replacing the excised lung and suffered no apparent respiratory embarrassment. From the facts cited in clinical experience, and the animal experimental evidence, it is obvious that the margin of safety is wide and that there is an ample excess of lung tissue to permit the removal of an entire lung without producing respiratory embarrassment following ordinary activity.

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RUSSELL TRACTION IN THE TREATMENT OF FRACTURES OF THE FEMUR*

OBSERVATIONS ON ONE HUNDRED FIFTY-SIX CASES

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IN LOOKING back over the evolution of the treatment of fractures of the femur, one is impressed by the rapid changes that have taken place. It is barely two decades since ice-tongs and plaster spicas were used extensively. It is even less since Steinman pins and Kirschner wires first made their appearance. When one considers the relatively short space of time that has passed between the popularity of each of these methods it would hardly seem necessary to urge that they have proven inadequate to obtain the results which we desired. Anyone who has had any experience in handling femur fractures can testify to the great difficulty that was frequently encountered in obtaining adequate reduction either by skeletal traction or by manual reduction and encasement in plaster. An infected knee joint or osteomyelitis was an occasional complication and always a source of worry. In a paper read before this Society in March, 1937, Kennedy,[26] reviewing a series of 120 cases treated by these methods, showed a high percentage of tong and pin wounds and general dissatisfaction with the use of plaster spicas. His conclusions seemed to be that some more efficient method should be employed in handling these fractures and that skeletal traction was far from the ideal solution.

Although Russell[1] suggested his method for treating fractures of the shaft of the femur in 1924, it was not until 1927 that Ryan,[2] of Philadelphia, brought the method to the attention of American surgeons. Published reports on the results obtained with this method have not been as numerous as one might expect and to many who have adopted it at all extensively, there is a certain lack of understanding as to its rather apparently limited use. As late as January, 1937, Van Gorder,[23] reporting a series of 105 consecutive femur shaft fractures from the Massachusetts General Hospital, did not even mention the Russell method of traction, even though he admitted that their current method of treatment necessitated open reduction in 25 per cent of their cases. In several other recent reports on large series of femoral shaft fractures,[7, 22, 24] the authors, similarly, seem to have omitted this method from their armamentarium. Where the method has been used, however, it has been so enthusiastically endorsed that it is difficult to understand why the reports have been relatively so few.[3, 4, 5, 6, 8, 9, 11, 12, 13, 14, 15, 19, 20, 25] Several authors,

* Read before the New York Surgical Society, December 6, 1939. Submitted for publication November 6, 1939.

notably Sallick,[16, 18] and Impink and Lee,[17] have called attention to its value and have strongly urged its more general adoption.

The results of open reduction of shaft fractures may be excellent under ideal conditions and in certain hands, and one may not agree entirely with Russell's dictum that "there is only one circumstance that can justify such a surgical procedure—that is mechanical impediment to the reposition of the fragments." One must, however, agree with Sallick that, whereas, open fixation may be the ideal form of treatment, its routine use is, in most hospitals, quite impractical and not without danger. Van Gorder's[23] series of cases, where the operative work was performed in a well recognized hospital, showed an infection rate of 15.4 per cent, which the author himself notes is deplorably high. Acknowledging that the infection rate might usually be expected to be much lower than this, it seems to be generally accepted that the routine use of open reduction and internal fixation would, in the majority of institutions, not be feasible. Weil, Kuehner and Henry,[24] reporting 103 operated cases, had five infections, with two deaths from septicemia, an infection rate of 4.8 per cent.

Because of our lack of satisfaction with the results that we were obtaining in femoral shaft fractures by means of skeletal traction, the Russell method was adopted on the Fourth Surgical Division of Bellevue Hospital in January, 1930. During the past five years this method has been used routinely for all fractures of the femoral shaft together with intertrochanteric fractures, but excluding fractures of the femoral neck that could be handled by any other means. Our routine of treatment has not differed materially from Russell's original description, except in some rather minor details. Russell's original article is still the best reference, as most of the subsequent commentators agree that, for clarity and brevity, his description cannot be improved upon. A brief comment upon the method as we use it, together with the mechanics involved, will be given later in this report. From January 1, 1930, to July 1, 1939, we have treated 156 patients by this method and feel that this is a sufficient number to justify an analysis of the method, together with some conclusions as to our end-results. There was only one compound fracture in this series. We have not included any cases in which the treatment was interrupted because the patient was unmanageable or where a plaster encasement was applied to permit transportation home or to another hospital.

In studying this series, the cases have been subdivided according to the location of the fracture, as shown in Table I.

TABLE I

LOCATION OF FRACTURE

Intertrochanteric	85
Upper one-third of shaft	23
Middle one-third of shaft	18
Lower one-third of shaft	30
Total	156

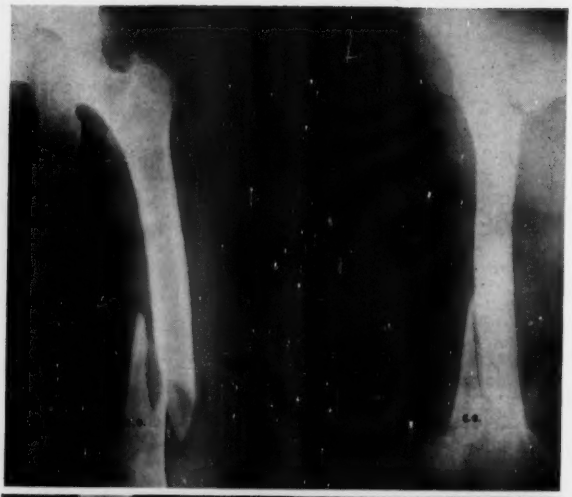

FIG. 1.—Case E. C.: Prereduction. Fracture lower third of shaft.

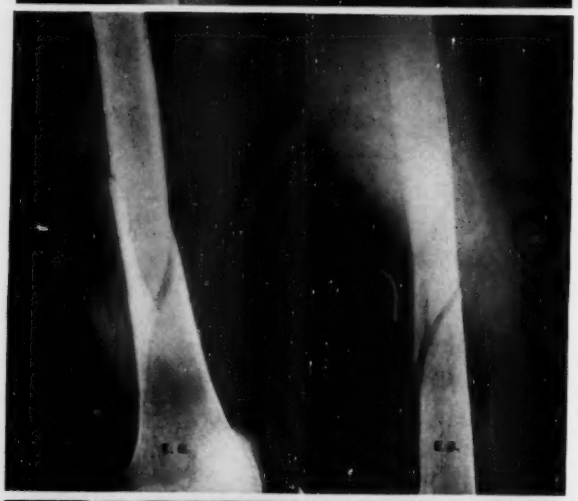

FIG. 2.—Case E. C.: Postreduction. After one week in traction.

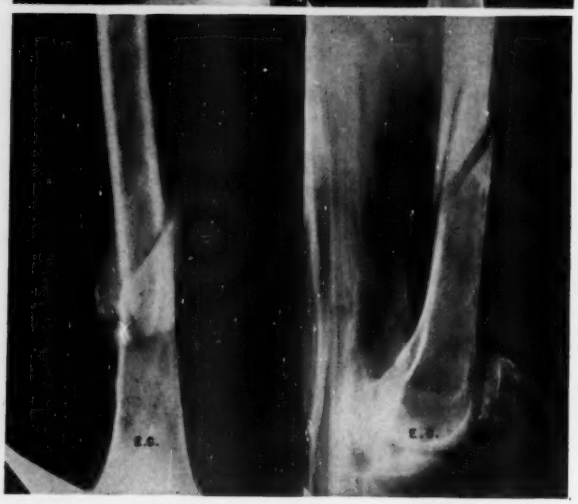

FIG. 3.—Case E. C.: Union after nine weeks in traction.

FIG. 4.—Case W. S.: Preduction. Fracture middle third of shaft.

FIG. 5.—Case W. S.: Postreduction. One week.

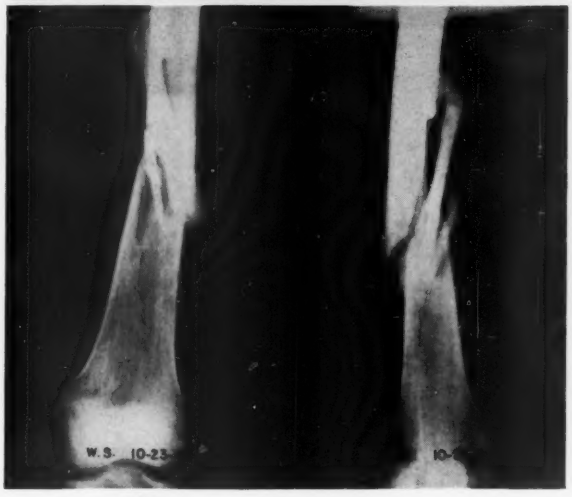

FIG. 6.—Case W. S.: Union after 16 weeks in traction.

In this group of 156 cases, there were seven cases in which adequate reduction could not be obtained. Three of these were in the upper third of the shaft, and four of them in the lower third. One of the patients whose fracture was in the upper third was treated early in our experience with Russell traction and because an adequate reduction could not be obtained it was decided to substitute skeletal traction by means of a Kirschner wire through the condyles. Following this procedure an infection developed which rapidly spread to the knee joint. The patient became desperately ill and, as a life saving measure, a midthigh amputation through the fracture site was performed. A second case in which the method did not give a satisfactory reduction was a patient with a compound fracture of the lower third of the shaft. The wound in this case was carefully débrided but was not closed, and Russell traction was applied. The characteristic posterior displacement of the lower fragment was present and could not be overcome and again, as in the first case, skeletal traction with a Kirschner wire through the condyles was undertaken. The position was much improved, but still not good, and open reduction was being considered. At this time, however, a severe infection developed in the compound wound, postponing all further attempts at reduction, and necessitating an amputation some eight days later. At this time muscle interposition was found between the fragments. Following the amputations, both the patients made satisfactory recoveries. It would seem as though open operation with internal fixation of the fragments would have been a better second choice than skeletal traction in these two cases, and such has since been our routine when Russell traction has not proven adequate. The five other cases all had open reductions with plating of the fragments, at which time it was found that there was muscle interposition in every instance.

The patients were all adults, of whom 74 were men and 82 were women. The youngest was 14 years of age and the oldest 86. The average age was 58. The better general condition of the patients and the ease of handling them, when compared to a similar group treated by skeletal traction, cannot be overemphasized. Most of the patients of advanced age have stood this method of treatment without complication even though traction was maintained in many cases for over 100 days. The patients were all kept in traction until firm bony callus had formed, enabling the patient to get out of bed with the aid of crutches or of an ambulatory Thomas splint. The shortest period that traction was necessary was 42 days and the longest was 135 days. The average period was 78 days.

The weight used for traction varied from five to ten pounds depending upon the muscular development of the patient and the amount of overriding, but in no case was more than ten pounds needed to obtain adequate reduction. In practically all instances five pounds of weight was all that was required to maintain reduction after shortening had been overcome. The estimation of the correction of shortening has always been dependent upon roentgenologic

observation of the fragments and not upon external measurement. The latter method is not as exact and is apt to give rise to many errors.

There were six deaths, a mortality rate of 3.2 per cent, and these were all in aged individuals. The cause of death in each case was the same, *i.e.*, senility plus terminal bronchopneumonia, the patients ranging in age from 70 to 82 years. No deaths occurred in the cases operated upon.

TABLE II

DAYS IN TRACTION AND DURATION OF HOSPITAL STAY

Maximum days in traction	135.0
Minimum days in traction	42.0
Average days in traction	78.0
Maximum days of hospitalization	235.0
Minimum days of hospitalization	52.0
Average days of hospitalization	100.7

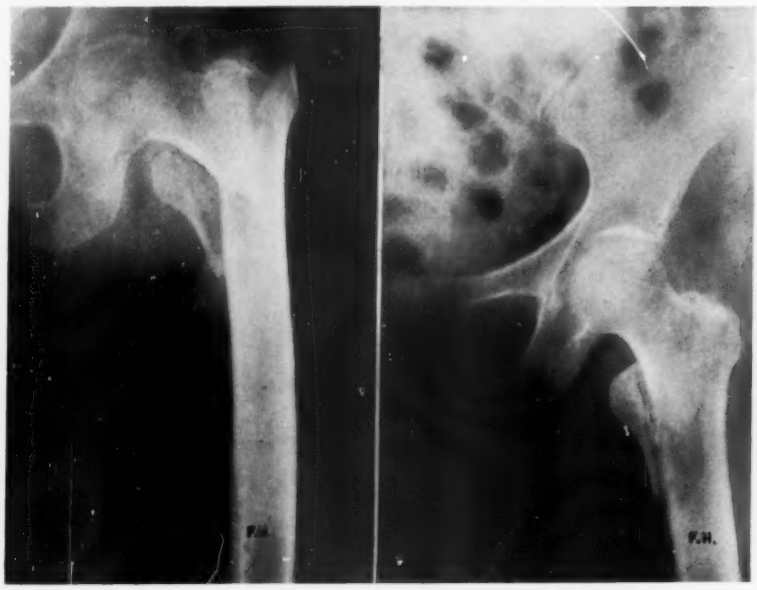

FIG. 7.—Case F. H.: Pre- and postreduction. Intertrochanteric fracture.

An accurate study of the time at which callus first appeared hardly seemed indicated as this is such a variable factor at all ages that it would seem as though no helpful conclusions could be drawn therefrom. For instance, one patient, age 82, with a lower third shaft fracture, showed exuberant bony callus at six weeks (Figs. 1, 2 and 3), whereas a young adult male, age 35, showed only very limited callus at 16 weeks (Figs. 4, 5 and 6). In both instances reduction was excellent and there was good bony contact between the fragments.

Evaluation of Results.—Of the 156 cases treated, seven could not be adequately reduced, and open reduction with Lane plating was performed in

five, with the regrettable amputations above cited in the other two. Six other patients died during the course of treatment, leaving 143 patients treated by Russell traction alone, who could be followed to an end-result. Of these, 77 had intertrochanteric fractures, 21 had fractures of the upper third of the shaft, 17 had fractures of the middle third of the shaft, and 28 had fractures of the lower third of the shaft. In all of these 143 cases reduction was readily accomplished and overriding corrected (Figs. 7 and 8). Union was obtained in all instances, and all patients were kept in traction until union could be demonstrated both by clinical examination and by the appearance of sufficient callus roentgenologically.

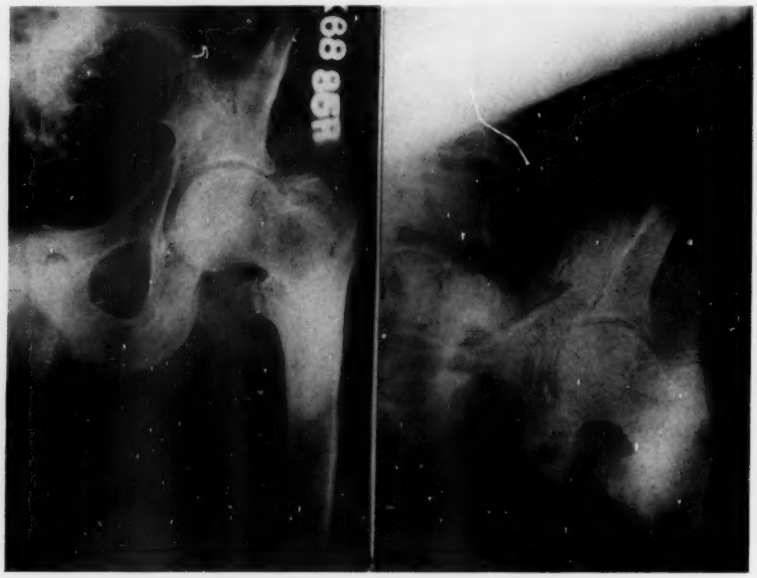

FIG. 8.—Case J. M.: Pre- and postreduction. Intertrochanteric fracture.

In the intertrochanteric group it has been our experience, however, that the demonstration of union is occasionally quite difficult. In the patient with the fat thigh, clinical examination is often of little value, and we have seen a number of cases in which the production of callus was quite limited. The surgeon's judgment as to when such a patient may be safely removed from traction is frequently taxed to the utmost. In our series of 77 intertrochanteric fractures, there were five patients who developed shortening of up to one inch subsequent to their removal from traction. Whether this was due to too short a period of traction, or whether the patient bore weight too early, it is difficult to ascertain. We do feel, however, that few femur fractures should be allowed unsupported weight bearing for at least six months from the time of the injury and this time will frequently be longer in the individual case.

In the shaft fractures, Russell traction has worked admirably except in

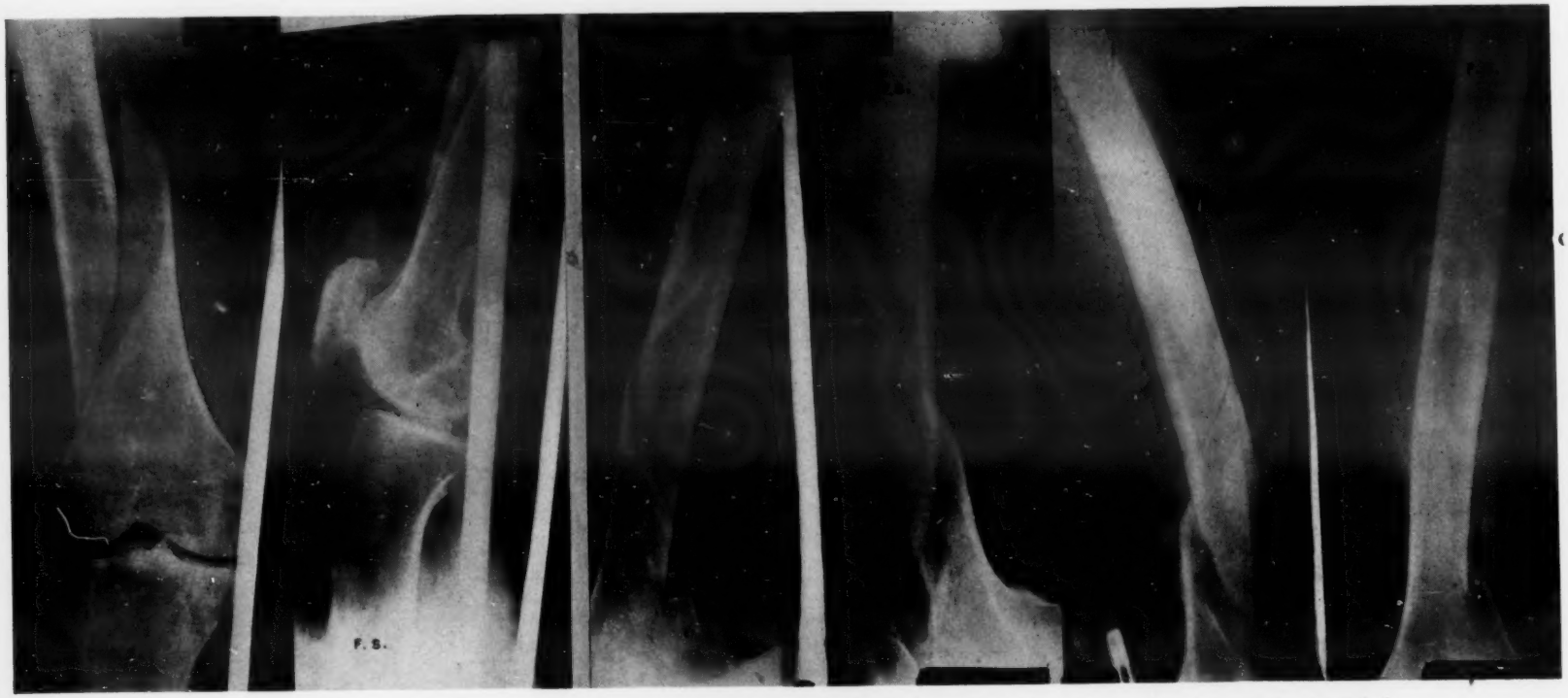

FIG. 9.—Case F. S.: Prereduction. Fracture lower third of shaft.

FIG. 10.—Case F. S.: Postreduction. One week and ten weeks, respectively.

the seven patients mentioned above. The ease with which displacement has been overcome has frequently been a source of extreme satisfaction, and we have been particularly fortunate in our results with the lower third fractures, where previous observers have noted difficulty in obtaining adequate reduction in many cases. The tendency of the lower fragment to be displaced posteriorly has been noticed with much less frequency since it has become routine practice in New York to apply Thomas splints with temporary traction at the site of the accident (Figs. 9 and 10). The Russell traction is instituted as soon as the patient is admitted to the hospital, regardless of the time of day or night, as it has always been our feeling that all fractures should be treated as surgical emergencies. Whether the early institution of traction before transportation of the patient to the hospital is a deciding factor in preventing the characteristic posterior displacement in this lower third group, we do not know. We do, however, seem to have had less difficulty in handling this group by the Russell method than had been the case in previous reports. If the Russell traction fails to give a good reduction within 24 to 48 hours, gentle manipulation under light anesthesia, without removing the traction, is suggested. We have become so impressed with the efficaciousness of the Russell method in shaft fractures that it is our feeling that, where reduction cannot be secured, one is usually justified in assuming that muscle interposition is present. In these cases open operation and internal fixation with a Lane plate is advocated.

One patient of particular interest was a female, age 39, who had a fracture of the shaft of both femora. She was handled very satisfactorily by the Russell method, the traction being applied to both lower extremities at the same time. She was in extreme shock for several days and any other method of treatment would have considerably added to the hazards of her recovery. Skeletal traction on both extremities would undoubtedly have been impractical as it would have been almost impossible to have kept from pulling her out of bed. No other instance has been brought to our attention where this method has been used on both limbs simultaneously.

There was no shortening in any of the shaft group, nor did any occur subsequent to the removal of traction. Callus here is usually more exuberant than in the intertrochanteric group, and clinical bony union is much easier to ascertain.

The restitution of joint function in these cases was most rapid and gratifying, and the period of after-care and after-treatment was considerably shorter than in previous patients where incorporation in plaster or skeletal traction was used. The prolonged period of stiff and swollen knees was not seen, and, indeed, joint function, up to a certain point, was always taking place in the hip, knee and ankle even while the traction was on. This was of particular value in the older group, where, as we all know, complete return of joint function previously implied long, tedious effort on the part of the surgical staff and considerable suffering on the part of the patient. Many of

the patients, when treated by the older methods, never did recover entire range of motion in the knee.

TABLE III

SUMMARY OF RESULTS

Total Cases			156
Shafts	71		
Intertrochanterics	85		
Method Unsatisfactory		7	
Open reduction-Lane plate	5		
Amputation (one compound fracture and one Kirschner wire infection)	2		
Deaths		6 (3.2%)	
Total Cases Treated Throughout by Russell Traction			143
Shafts	64		
Good results		64	
Intertrochanterics	79		
Good results		74	
Unsatisfactory results (shortening up to 1 inch developing after traction was removed)	5		
Total Good Results			138

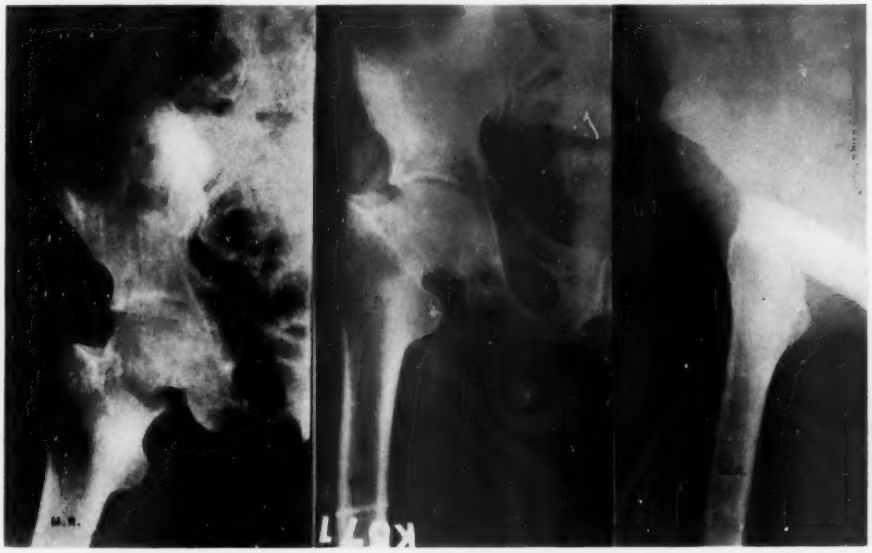

FIG. 11.—Case M. R.: Intracapsular fracture of the neck of the femur. The only case of the type in which union was obtained.

We had the opportunity of observing the efficacy of this method in a series of intracapsular fractures of the neck of the femur in a group of 18 aged individuals in whom other, more appropriate methods of treatment could not be used. These patients were all very senile or were suffering from physical complications of such severity as to make the use of the Smith-Petersen nail out of the question. Four of the patients refused operation

and were placed in Russell traction purely as a palliative treatment. Of the group of 18, five died while in the hospital, the date of death ranging from a few days to six weeks following admission. Two succumbed to senility and malnutrition and the other three to pneumonia. Of the total number, union was obtained in only one case, which occurred in a male, age 62, who refused to have the Smith-Petersen nail used and who was left in Russell traction, without any real hope on our part of his obtaining union (Fig. 11). The fragments were not impacted, there being a small amount of separation, but, even so, union ultimately occurred. We definitely feel that Russell traction should not be used as a method of treatment for fractures of the femoral neck. It is our experience that its use should be limited to that of a palliative method where the fracture has to be disregarded because of the age or physical condition of the patient.

TABLE IV

RESULTS IN INTRACAPSULAR FRACTURES OF THE FEMUR TREATED BY RUSSELL TRACTION

Total cases	18
Deaths	5
Nonunion	12
Union	1

Consideration of the Method Itself.—It is not the purpose of this paper to go into a detailed description of the Russell method or to discuss the mechanics involved. The original description by Russell is still as clear and concise as can be found in any of the succeeding reports, and no improvements on his original description have been brought forth that are of fundamental importance.[4] The routine which we have followed may be briefly summarized as follows:

(1) The foot of the bed is elevated six to eight inches.

(2) The thigh is elevated by a pillow underneath, so that the angle between the thigh and the bed will be somewhere between 15° and 20°.

(3) The overhead pulley should be in a position some place between the tibial tuberosity and the middle of the leg, and the rope running from the sling to the overhead pulley should make a right angle with the leg. The overhead pulley, however, should never be so far below the knee as to cause the sling to slip out of position.

(4) The knee should be slightly flexed, so that the leg will make an angle of 10° to 15° with the bed or, in other words, so that the angle between the knee and the thigh lies between 150° and 160°.

(5) The extremity should not be abducted more than 15° from the midline.

(6) The leg should be shaved before the moleskin is attached, and the moleskin should be allowed to set for at least 20 minutes before any weight is attached.

(7) The head of the bed should never be "gatched-up" while the patient is in traction, although a pillow may be allowed under the head.

(8) Rope should be as small in caliber as can be obtained and pulleys large, although, practically, we have not found that, within reasonable limits, this has made any essential difference in the efficacy of the apparatus.

(9) The weight applied should vary from five to ten pounds. The weight should be immediately reduced if any evidence of overpulling is seen roentgenologically.

(10) A portable roentgenographic apparatus is essential and roentgenograms should be taken every day or two for the first few days, so that the position of the fragments may be constantly checked and overpulling avoided. Overpulling of the fragments should be particularly guarded against, especially in transverse fractures. Blum[27] has pointed out the frequency with which delayed union is associated with this condition.

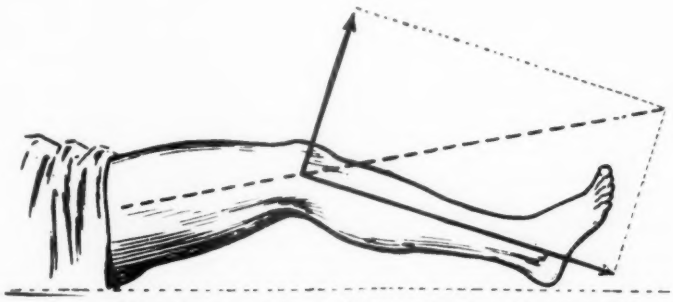

FIG. 12.—Mode of action of the two forces employed. (After Russell.[1])

If the above suggestions are followed, we feel that the mechanics of the method will be automatically taken care of. The line of the femur will become the resultant of the parallelogram formed on one side by the leg and on the other side by the rope running from the sling under the knee to the overhead pulley (Figs. 12 and 13). Mathematically calculated, it has been constantly found that the pull exerted along this resultant and, therefore, along the long axis of the femur is somewhat more than twice the amount of the weight used for traction. The physical and mathematical calculations governing the mechanics of the force exerted along the resultant of the parallelogram constructed by the Russell method are quite complicated and require trigonometric equations to solve them. A complete understanding of these is not at all essential to an adequate appreciation of the method or ability to handle it, and the reader is referred to three excellent articles by Ryan,[2] Lowry,[10] and Wilson[21] on the complete physics and mathematics involved.

The other aspect of the Russell traction method, namely, that of putting the muscles of the lower extremity in a position of physiologic rest by slight flexion at the hip and knee with the added support of the pillows, has not been sufficiently stressed. In this regard it has always seemed to us a trite comparison to note that when we are about to go to sleep the joints of the body are usually in a position of moderate flexion, with the one exception of

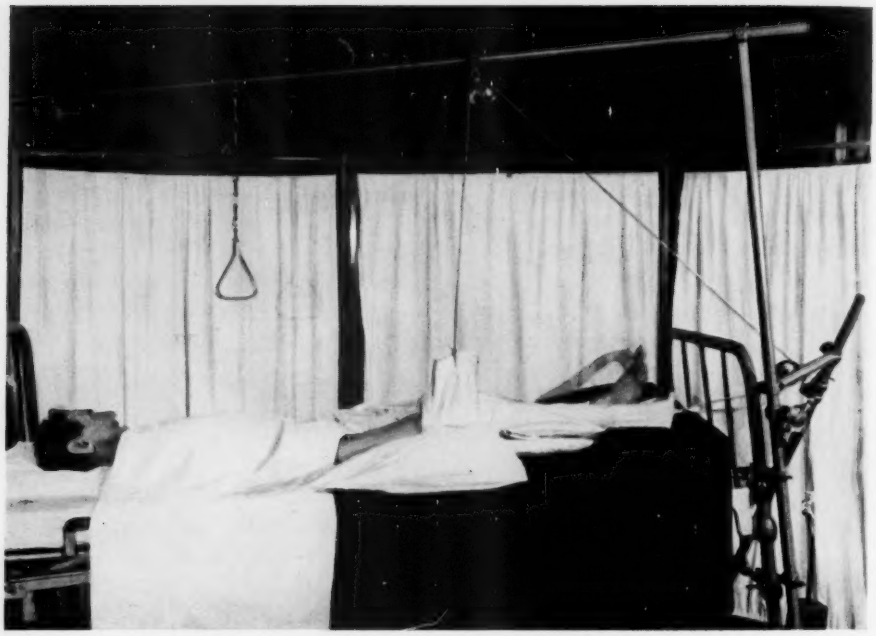

FIG. 13.—Russell traction properly applied.

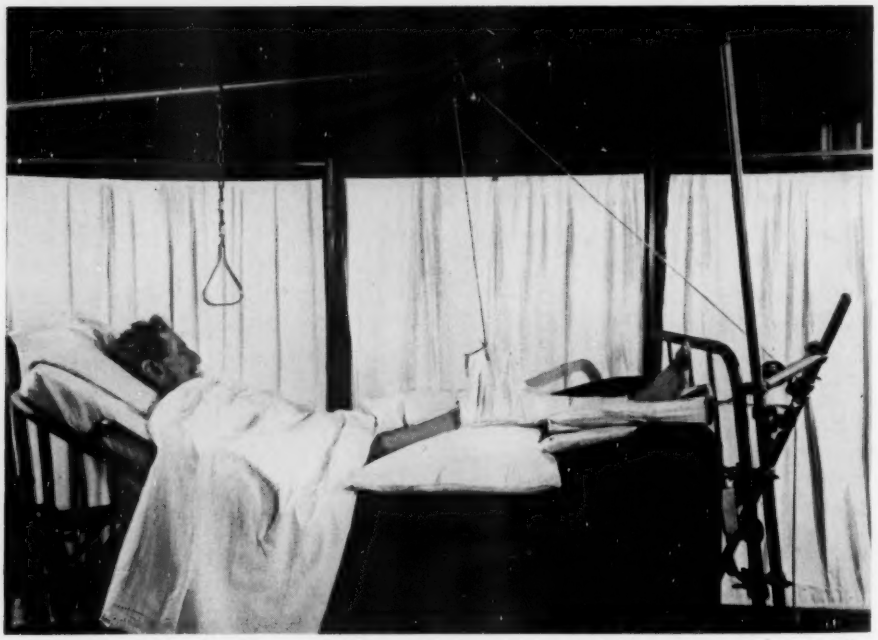

FIG. 14.—Russell traction improperly applied. Note the head of bed "gatched-up." Overhead pulley cephalad to knee. Patient slipped down in bed. Mechanics of method destroyed.

the ankle joint. This attitude of limited flexion, then, is probably the one of maximum physiologic relaxation for all muscle groups and, from this fact, we should learn a lesson in our management of fractures. Russell was the first to point this fact out, and our experience with his method amply satisfied us that his observations were correct. If the muscle groups are relaxed to the maximum of our ability, the spasm and tonicity will be greatly lessened, with a consequent relaxation of the muscle pull which is responsible for most of the overriding and displacements in fracture fragments. One only has to observe the ease with which overriding of the femur fragments is usually accomplished by this method in comparison to the difficulties which were not infrequently encountered when skeletal traction was used. In the latter instance, the flexion at the hip was usually much more acute, due to the frequent high elevation of the Thomas splint, as was also the flexion at the knee, where the use of the Pierson extension was so popular. Occasionally, in these cases, even 35 or 40 pounds of traction would not completely correct the overriding, demonstrating that by merely increasing the pull on already traumatized muscles, one could not overcome their contraction. Contrasted to this, the Russell method shows what gentleness in handling muscle groups will accomplish.

Like any system of traction, however, this method requires constant supervision by someone familiar with the necessary essentials. This knowledge is not hard to acquire, but due regard must always be maintained to see that correct angles at the knee and hip are constant, that the overhead pulley is kept in its proper relationship to the upper end of the tibia and that the patient is not allowed to slide down in bed and remain there (Fig. 14). The nursing staff must be taught the proper position of the pillows and of the overhead pulley, so that, when sheets are changed and bed pans passed, the mechanics of the system will not be disturbed. If all of these simple suggestions are followed, one will find the method efficient and safe and eminently more comfortable for the patient than any other system of treatment that we know.

CONCLUSIONS

(1) One hundred fifty-six fractures of the femur treated by the Russell traction method are reported, with good results in 138 cases.

(2) It is the method of choice for intertrochanteric fractures and for all of those involving the shaft.

(3) If good reduction in shaft fractures cannot be obtained, and if gentle manipulation under an anesthetic will not improve the position of the fragments, it may be assumed that muscle interposition is present. Open reduction and internal fixation with a Lane plate is then advocated.

(4) The Russell method is not recommended for treating intracapsular fractures of the neck of the femur and should only be used here as a palliative measure. In 18 cases in which we used it, union was obtained in only one patient.

(5) Skeletal traction should be discarded as a procedure in handling femur fractures.

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DISCUSSION.—DR. ROBERT H. KENNEDY (New York) said that in spite of several papers having been published during the past ten years regarding Russell traction, the majority of surgeons are still unaware of its great usefulness, and that Doctor Lewis' paper is particularly valuable because it records his experience and results in by far the largest series yet published. Doctor Kennedy agreed with him in all major points.

In 1932, Doctor Kennedy and his staff at Beekman Street Hospital commenced using Russell traction as the preferred treatment in intertrochanteric and all femoral shaft fractures. In general, it has not been satisfactory in transverse fractures of the middle third and in the lower third fractures with posterior angulation of the distal fragment. In these, open reduction should be performed unless satisfactory position is being maintained at ten days to two weeks after the accident. The danger of overpull in the middle third is great and one-sixteenth inch distraction means many weeks' delay in union.

Russell traction is the best treatment at the present day for intertrochanteric fractures; it usually is efficient in upper third cases, and it is eminently satisfactory in oblique and comminuted fractures of the middle third. Its use for the treatment of a femoral neck fracture is inexcusable.

The method, as is true of all traction, requires the full cooperation of interns, nurses and orderlies. Doctor Kennedy said he mentioned orderlies, in particular, because the use of the bedpan is one of the easiest ways to have traction become disarranged. All must be trained by the visiting staff to know right from wrong and to know how far they can go in adjusting something they find is wrong. Any group of nurses, accustomed to caring for fracture patients treated by different methods, will admit that they prefer attending a case in Russell traction to any other method.

A footpiece should never be a part of the set-up. It subtracts an unknown amount of pull. Doctor Kennedy said he had never seen the slightest loss of any function at the ankle joint with Russell traction continued as long as four months, and that function at knee and hip returns more promptly with this than with other methods.

Manipulation under anesthesia while in traction should be used more frequently. If the desired position has not been attained in 24 to 48 hours such manipulation should be done rather than later.

Before the use of Russell traction, these fractures in children of six to 15 years of age were a source of great worry. Reduction followed by a plaster spica resulted, too frequently, in later slipping with overriding. The child is too large for Bryant traction. Adhesive plaster traction with suspension is not at all satisfactory. One hesitates to put in ice tongs or Steinman pins because of possible interference with the epiphyseal line. Kirschner wires are less dangerous in this regard. Open reduction hardly seems proper, except for muscle interposition, when practically all these fractures heal anyway, but no one likes to care for a case remaining in malposition. Russell traction has happily solved the problem in most instances in these children. Doctor Kennedy could not agree with Doctor Lewis that skeletal traction should be entirely discarded in fractures of the shaft of the femur.

Russell traction is useful for a number of other purposes, *e.g.*, fracture of the acetabulum with or without central displacement of the head of the femur, fracture of the pelvis with displacement of fragments, immobilization following open reduction of fracture of the femur, and immobilization following nailing of the fracture of the neck of the femur.

As used in many hospitals this treatment is merely a name and a travesty on the method suggested by Russell.

DR. A. STILLMAN, 2ND (New York), said that he and the staff at Roosevelt Hospital had been using Russell traction since 1931, having been stimulated to adopt it by Doctor Grace's success at Bellevue. So quickly did the house staff catch on to the principle, and so easy was it to apply, that practically every fractured femur, whether of the neck or shaft, whether old or young, was put up in this traction, and fairly promptly.

With other methods this early application of treatment did not obtain. The use of tongs, pins, wire or even a plaster encasement required the presence of an attending surgeon and this made for delay. With regard to Doctor Lewis' suggestion that the bettering of results in the lower third fractures may be due to the ambulance surgeon's following the dictum of the American College of Surgeons' Fracture Committees to "splint them where they lie," Doctor Stillman said that certainly this earlier treatment in the hospital is one factor. A second factor is the lessened manipulation or monkeying with the fracture after it is put up as compared to other methods. The patient is so comfortable, so relaxed, the reduction is earlier accomplished, and then the patient is let alone. Constant motion at the fracture site is a recognized cause of delay in union.

Of delayed union, Doctor Lewis was fortunate to have none, but it still occurs as evidenced by two cases in recent years at Roosevelt Hospital. One, after 68 days, showed almost no sign of callus, and so a plaster encasement was applied, and after another 60 days was plated, and became solid in seven months. The other case was an upper third fracture in a heavy young man. After Russell traction failed, it was plated, the bone became infected, the plate was removed and now, after two years, is not solid. However, Doctor Lewis' experience must mean that delayed union is less common by the Russell traction method.

In regard to stiffness of the joints, much work must yet be done to accomplish full motion, but the traction pull being through the knee joint capsule gives a better start than in other methods. Doctor Stillman said he had performed one quadriceps extensor lengthening for a stiff knee. The ankle joint does not get stiff because the foot can be, and is, moved by the patient all the time. Doctor Stillman has had little trouble with the hip because, he feared, these patients are gatched-up for their meals. Doctor Lewis warns against this, but does it make an important difference?

Doctor Lewis had 77 intertrochanteric fractures with only five having shortening up to an inch. This is a very good average result. In a small group looked up by Doctor Stillman there were three in 12 cases. This is an older age-group, 72 in Doctor Stillman's series, and four out of 16 died. All six of Doctor Lewis' deaths seem to have been in this group.

Of the shaft, Doctor Lewis showed seven failures, or a little less than 10 per cent, again an exceedingly good record. Out of 12 cases on Doctor Stillman's service, four were plated because of poor position or delayed union—seven had a good result, and one an inch of shortening.

Doctor Lewis has no children under 14 on his service and so this group was not included in his presentation, but Russell traction works exceedingly well with them. Doctor Stillman said he had several times demonstrated the advantage of Russell traction over the Bryant vertical traction to his staff.

As to fractures of the neck of the femur, if Whitman's method of putting these cases up with the thigh extended and internally rotated is right, Russell traction with the thigh flexed and somewhat outwardly rotated must be wrong.

A member of Doctor Stillman's staff looked up these cases and found only one union.

Doctor Stillman concluded that, all-in-all, he favors the Russell traction method and was much indebted to Doctor Lewis for having provided a standard to shoot for.

DR. PHILIP D. WILSON (New York) said that he would always remember having met Mr. Russell, approximately ten years or more ago, at the time no one in this country knew of Russell, or Russell traction. He accompanied Doctor Wilson on his rounds of the Fracture Ward and fell into a discussion with him about treatment of fractures of the shaft of the femur. He asked Doctor Wilson what he was aiming for in the way of alignment, and Doctor Wilson replied that he tried to get as nearly end-to-end approximation as possible. Mr. Russell took the view that it was better to have the fragments in corner-to-corner approximation or even with a little overriding because he had observed that union occurred more quickly and more solidly than with end-to-end approximation. From the functional standpoint, he said that the result was just as good one way as the other and Doctor Wilson had to agree. Mr. Russell then went on, very modestly, to describe the method of traction that he was using. He made a diagram of it and made an analysis of the different forces that were brought to bear upon the fracture by the method of rigging. Doctor Wilson was impressed by the simplicity of the Russell system but it seemed preposterous that a weight of five to ten pounds could be made to do the same work that then required from 35 to 40 pounds of weight when attached to ice tongs or a pin to accomplish. It took several years of observation and experience to find out that all of Russell's claims were true.

Doctor Wilson said, however, that, much as he admired the efficiency and simplicity of Russell traction, he felt it unwise to stress it as the only method of treatment for fractures of the shaft of the femur. His practice is to allow surgeons to choose their own methods while holding them responsible for their results. At a recent meeting of the American Orthopedic Association there was a symposium on "Fracture of the Shaft of the Femur," with 10 or 11 different speakers, and almost everyone advocated a different method of treatment and presented results to substantiate what he claimed. Indications for these different methods of treatment should be formulated and some agreement reached about them.

Doctor Wilson was very much impressed by the results presented by Doctor Lewis but was not sure whether he was talking about end-results, that is, results seen and examined a year or so after discharge from the hospital, or hospital-results, because these are two different matters and analysis some time later will be quite different from that made at the time the patient is discharged from the hospital. The amount of shortening will be greater and there will be bowing and other complications. He asked Doctor Lewis if he would clarify this point, and in concluding complimented Doctor Lewis upon having made an excellent case for Russell traction.

DR. KENNETH M. LEWIS (closing) said that he realized his results with lower third shaft fractures had been better than those reported by Doctor Kennedy. He was unable to account for this unless it was due to the fact that these patients have all been coming into the hospital in Thomas splints with traction applied, the patient having been splinted at the site of the accident. He said he was not giving Russell traction credit for reducing these fractures because they did not have any posterior displacement when they arrived. If one considers, however, the acute angle at which the thigh was usually elevated when skeletal traction was used with a Kirschner wire through the con-

dyles and with the leg frequently acutely flexed on the thigh, one will admit that the muscles were certainly not in a position of physiologic muscle balance. Doctor Lewis said he had often wondered whether the posterior displacement of the lower fragment did not occur when these cases were put up into traction or whether the displacement did not get worse while they were in traction. His results with skeletal traction in the lower third when they had any posterior displacement have been very unsatisfactory.

Doctor Lewis said that when he stated skeletal traction should be discarded in the treatment of fractures of the femur, he realized it was probably too strong a statement to make. On the other hand, one must be guided by his own experience and Doctor Lewis' has been that by using Russell traction routinely the results had been much better. In those cases in which Russell traction does not give an adequate reduction skeletal traction is not even considered, but an open reduction is performed. Fortunately, there have not been any delayed unions in the shaft group.

Regarding end-results, Doctor Lewis said that the results he had reported might be classified as a combination of those cases followed in the wards and in the Follow-Up Clinic, plus those patients cared for while in the hospital but not seen thereafter. Many of the patients at Bellevue Hospital have no homes and so are kept there for relatively long periods of time, until union both by roentgenologic examination and clinically is complete. Thus there is an opportunity for rather long periods of observation and follow-up, even before the patient leaves the hospital. They are not allowed to bear any weight without the aid of an ambulatory splint of one kind or another, unless they are able to handle crutches adequately. Doctor Lewis felt that as far as function in the joints is concerned the results from Russell traction show a big improvement over those with plaster or skeletal traction. Concerning bowing afterwards, it seemed to Doctor Lewis that if there is firm bony callus in a shaft fracture, and that shaft is protected with a walking caliper, and the patient is not allowed to bear any unsupported weight for at least six months, there can be no excuse for the occurrence of bowing. With some of the femur cases, this period of nonweight bearing should be extended to eight or nine months.

TOTAL THYROIDECTOMY FOR HEART DISEASE*

A FIVE-YEAR FOLLOW-UP STUDY

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TOTAL THYROIDECTOMY for heart disease was first performed in 1932. This surgical procedure represented an attempt to bring relief to cardiac derelicts by altering the normal physiologic mechanism. The rationale of this procedure has been repeatedly discussed in publications from this clinic and elsewhere.[5, 11, 12, 19] Numerous reports have presented the immediate and early results, the technic of the procedure, and the fundamental changes which are brought about.[1, 4, 8, 9, 10, 13, 20, 21, 22, 23, 24, 25] Sufficient time has now elapsed since total thyroidectomy for heart disease was first performed for a more considered opinion than has heretofore been available. In this report we are including every patient submitted to total thyroidectomy for heart disease in the Peter Bent Brigham Hospital during 1932, 1933, and 1934. Either the present status or cause of death has been ascertained.

The cases in our study total 57, and have been divided into two fundamental groups: Those (32 patients) with intractable angina pectoris; and those (25 patients) with some form of congestive heart failure which did not yield to the usual conservative measures. Sixteen patients survived a five-year period, 12 in the angina pectoris group and four in the group with congestive failure (Table I); all but one of these were personally examined by us.

TABLE I

MATERIAL

	Number of Operations	Patients Living November, 1939
Angina pectoris	32	12
Congestive failure	25	4
	57	16

Most of the patients who did not survive were closely followed in the hospital dispensary, so that the records show the extent and duration of any improvement that may have taken place after operation. The data concerning the cause of death in the 41 nonsurvivors are based on autopsies in 14 instances, and on observations made in our wards in seven cases where death occurred in this hospital but necropsy was refused. The data on the remaining 20 cases were compiled from letters from attending physicians or death certificates (Table II).

* Read before the American Surgical Association, May 1, 2, 3, 1940, at St. Louis, Mo.

TABLE II

SOURCES OF FOLLOW-UP DATA

	Total No. of Cases
Five-year survivors	16
Office visit	13
Home visit	2
Telephone call	1
Deaths	41
Autopsy	14
Died in P.B.B.H.; autopsy refused	7
Death certificate	11
Letter from family physician or other hospital	9

During the first year following operation 11 of the 25 patients with congestive failure succumbed, in contrast to seven of the 32 with angina pectoris (Table III). In the succeeding years the mortality in each group did not

TABLE III

PERIOD OF POSTOPERATIVE SURVIVAL

	Total Patients	Living at End of 1 Year	2 Years	3 Years	4 Years	5 Years
Angina pectoris	32	25	22	18	15	12
Congestive failure	25	14	11	8	6	4
	57	39	33	26	21	16

exceed four deaths per year. There were five postoperative deaths* in the 57 cases, and all but one, thought to be due to pulmonary embolism, could be directly attributed to heart disease. This mortality rate agrees closely with the operative mortality of 9 per cent reported from the Beth Israel Hospital, Boston, which has had the largest experience with total thyroidectomy for heart disease,[3] but it is somewhat higher than that noted in the collected statistics from other clinics.[13, 21, 24] However, the operative mortality in general is surprisingly low when one considers the poor surgical risks these patients present. As one would expect, nearly all of these patients die eventually from their fundamental cardiac disorder (Table IV). Total

TABLE IV

CAUSES OF DEATH

	Total Deaths	Post-operative	Deaths Attributable to Heart Disease	Other Causes
Angina pectoris	20	3*	16	1 (cerebral hemorrhage)
Congestive failure	21	2‡	17	2 (pneumonia without congestive failure)
	41	5	33	3

* Coronary thrombosis, one day p.o.
Pulmonary edema, one day p.o.
Coronary thrombosis, five days p.o.
‡ Coronary thrombosis, day of operation
Pulmonary embolism, one day p.o.

thyroidectomy should not be regarded as more than a special therapeutic attack upon a group of diseases at present incurable and ultimately fatal.

* In the postoperative death group we include all who died in the hospital within one week of the surgical procedure.

An analysis of the results in each case, on the basis of relief of symptoms, leads to further contrasts between the group with angina pectoris and the group with congestive failure (Tables V, VI, and VII). Despite the small

TABLE V

RELIEF OF PAIN IN ANGINA PECTORIS

A. Patients surviving less than six months		5
B. Patients surviving six months to five years		15
Relief for six months or more	14*	
No relief at six months	1	
C. Patients surviving more than five years		12
Marked and sustained relief (patient's estimate) (substantiated by greater activity and less medication for pain—six)	8	
Temporary relief for six months to two years (degree variable, unimproved at present)	4	
No relief at any time	0	
		32

* In some instances had relief almost to the time of death.

TABLE VI

RELIEF OF CONGESTIVE FAILURE IN CHRONIC RHEUMATIC VALVULAR DISEASE

A. Patients surviving less than six months		5
B. Patients surviving six months to five years		7
Clinical improvement for six months or more (increased activity, fewer symptoms)	5	
No definite improvement	2	
C. Patients surviving more than five years		4
Sustained clinical improvement	1	
Improved for two years	1	
Improved for more than five years, until death from congestive failure	2	
		16

TABLE VII

RELIEF OF CONGESTIVE FAILURE IN ARTERIOSCLEROTIC OR HYPERTENSIVE HEART DISEASE

A. Patients surviving less than six months		5
B. Patients surviving six months to five years		4
Moderate or marked clinical improvement for six months to three and one-half years	3	
No definite improvement	1	
C. Patients surviving for more than five years		0
		9

number of cases certain trends appear clear. In the angina group, with a median age of 61, there are 12 who have survived five years, eight of whom have had sustained clinical improvement. In the congestive failure group, with a median age of 44, only four have survived five years, three of whom have had sustained clinical improvement.* This result is in close accord with the early experiences of other clinics, where the most favorable results have occurred usually in the group with angina pectoris.[13, 21, 24] This may be brought out in another way. Of the 27 patients with angina who lived six months or longer, 26 were at least partially relieved for periods longer than six months. By way of contrast, only 12 of the 15 patients with congestive failure who lived six months or longer were at least partially improved for periods longer than six months.

If the group of patients with congestive failure is subdivided as in Tables VI and VII, one sees an improvement in outlook for those with congestive

* Two of these have died of congestive failure during their sixth postoperative year.

failure from rheumatic valvular disease. However, the slight improvement does not justify much optimism. The median age in this group is 40—20 years less than in the angina group—but the life expectancy in patients with mitral stenosis with marked decompensation is very short. Moreover, it is difficult to evaluate improvement in a group of patients who suffer from recurrent decompensation. Apparent improvement may be merely a natural remission in the disease, and one is less justified in carrying out a radical procedure unless *sustained* improvement is to be expected. We are inclined to be rather pessimistic about the entire congestive failure group. From our small experience with nine cases of congestive failure from arteriosclerotic and hypertensive heart disease, not one of whom survived five years, we feel that total thyroidectomy will give disappointing results if employed here. In the rheumatic group with congestive failure there may be a place for the operation—we have had several cases where we felt there was prolonged and definite benefit—but, unfortunately, there does not appear to be any way to tell in advance which patients will do well. In view of the uncertain benefits and the limited life expectancy, we no longer perform total thyroidectomy for congestive failure.

Our best results from total thyroidectomy have been obtained in the group of patients with intractable angina pectoris. It is admittedly difficult to evaluate improvement in a purely subjective phenomenon like pain, and it is well for us to bear in mind that angina pectoris may undergo remissions and exacerbations like any other chronic disease. It may even cease spontaneously. A well-known tendency under such circumstances is to ascribe the natural improvement to the therapeutic measure employed at the same time. Patients with angina may learn to avoid activities producing pain, and thus bring about fewer attacks. Sometimes the original diagnosis may be in error, as questioned in one of our cases (R. H.). Although our series is numerically small, the relief of pain following total thyroidectomy appears to be beyond question. Every one of our patients, living longer than a few days, had at least temporary improvement. In some cases the relief has been enduring; in others it has lasted only a few months before the reappearance of angina—usually, but not invariably, milder and of a different character. No agreement exists as to the mechanism by which relief is obtained.

The question is raised whether life is prolonged in patients relieved of their pain by total thyroidectomy. Since the prognosis for life varies so widely, a much larger series of cases than is included in the present study will be required to settle the problem. In one recent study of prognosis in angina pectoris the duration of life varied from one month to 23 years after the onset of symptoms.[15] In this same study no signficant change in prognosis could be demonstrated between those who had angina decubitus and those who had it only on effort. In the much more serious group, who have had coronary thrombosis, Dublin[14] cites statistics from several sources showing that patients surviving a first attack may live for years; 28 per

cent in one series survived five years or longer, 20 per cent in another. Our small series is without statistical significance on this point.

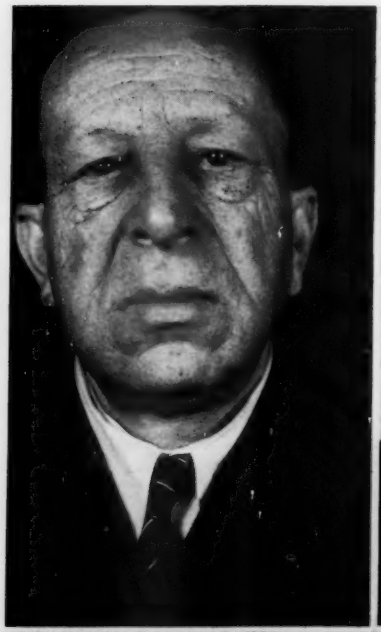
It is likewise difficult to select patients who are suitable for total thyroidectomy in the angina pectoris group. Patients making up this series were selected largely on the basis of intractability of pain to ordinary therapeutic measures. Many of them had angina decubitus (ref. Case summaries). No patient in the group of 12 five-year survivors had frank signs and symptoms of congestive failure before operation, while six in the group of 20 who did not survive gave definite evidence of congestive failure. Three of the five-year survivors had some cardiac enlargement before operation—in two it was only slight. Ten of the nonsurvivors had cardiac enlargement—in six this was slight. From this it would appear that either congestive failure or cardiac enlargement is an unfavorable prognostic sign. Interestingly enough, a previous coronary thrombosis does not seem to affect the prognosis adversely.

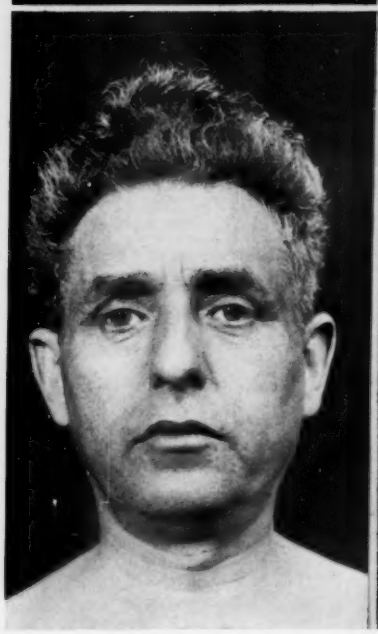
It is noteworthy that in the five-year follow-up the problem of myxedema in these thyroidectomized cardiacs does not loom large. In no instance in the entire series has myxedema offered a problem comparable to the heart disease for which operation was undertaken. Several patients (S. G., with spontaneous myxedema before operation, and G. S., who was psychotic and uncooperative) have offered considerable difficulty; the remainder, however, almost without exception have proved amenable to management. The case summaries of the survivors and the photographs bear out this point. Nearly all of the surviving patients feel the cold easily, and some of them speak slowly. Although most of them believe they are slowed down mentally, we have difficulty in attributing this possible change to the imposed myxedema but see in it rather the normal slowing down with age. As can be seen in the case summaries, there is considerable variation in the amount of thyroid taken by different patients. We regard these patients, like diabetics, as continuous problems in management, and an effort is made to explain this to them, to see them at regular intervals, to check the basal metabolic rate as needed, and to adjust the dosage of thyroid to the optimum for each individual patient. Should the basal metabolic rate become elevated, there may be a recurrence of angina symptoms. It is very important to bear in mind not only that the optimum dose of thyroid extract may vary with each patient but also that in any individual this requirement may vary from time to time. In our experience a basal metabolic rate of about −15 was satisfactory in the majority of patients, but as the case summaries reveal this level cannot be utilized as the optimum level for all patients. Our experience agrees with others, that surgical myxedema need not interfere too much with the patient's enjoyment of life nor become a serious problem in control.[3, 10, 13]

SUMMARY AND CONCLUSIONS

We are able, at this time, to report a five-year follow-up of 57 consecutive cases of total thyroidectomy performed for heart disease during 1932, 1933,

FIG. 1. FIG. 2.





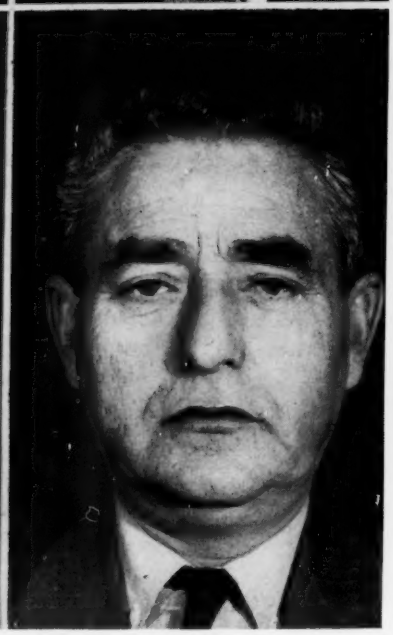


FIG. 3A. FIG. 3B.

FIG. 1.—M. G., age 61. November, 1939. Six years and two months after operation. B.M.R. +5.

FIG. 2.—W. D., age 60. November, 1939. Five years and 10 months after operation. B.M.R. −11.

FIG. 3A.—B. F., age 50. April, 1934. Before operation. B.M.R. +13.

FIG. 3B.—B. F., age 55. November, 1939. Five years and seven months after operation. B.M.R. −14.

FIG. 4.

FIG. 5A.

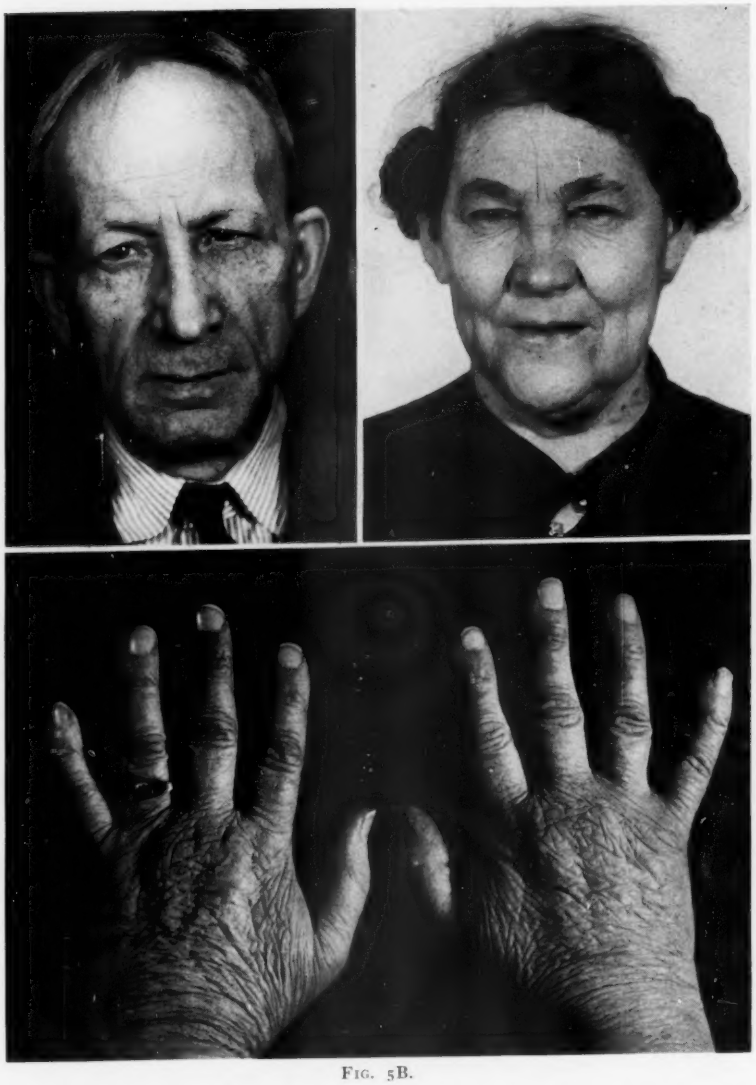


FIG. 5B.

FIG. 4.—H. Z., age 61. December, 1939. Five years and nine months after operation. B.M.R. −15.

FIG. 5A and B.—S. G., age 55. November, 1939. Five years and seven months after operation. B.M.R. −16. Spontaneous myxedema preceded operation.

FIG. 6. FIG. 7.

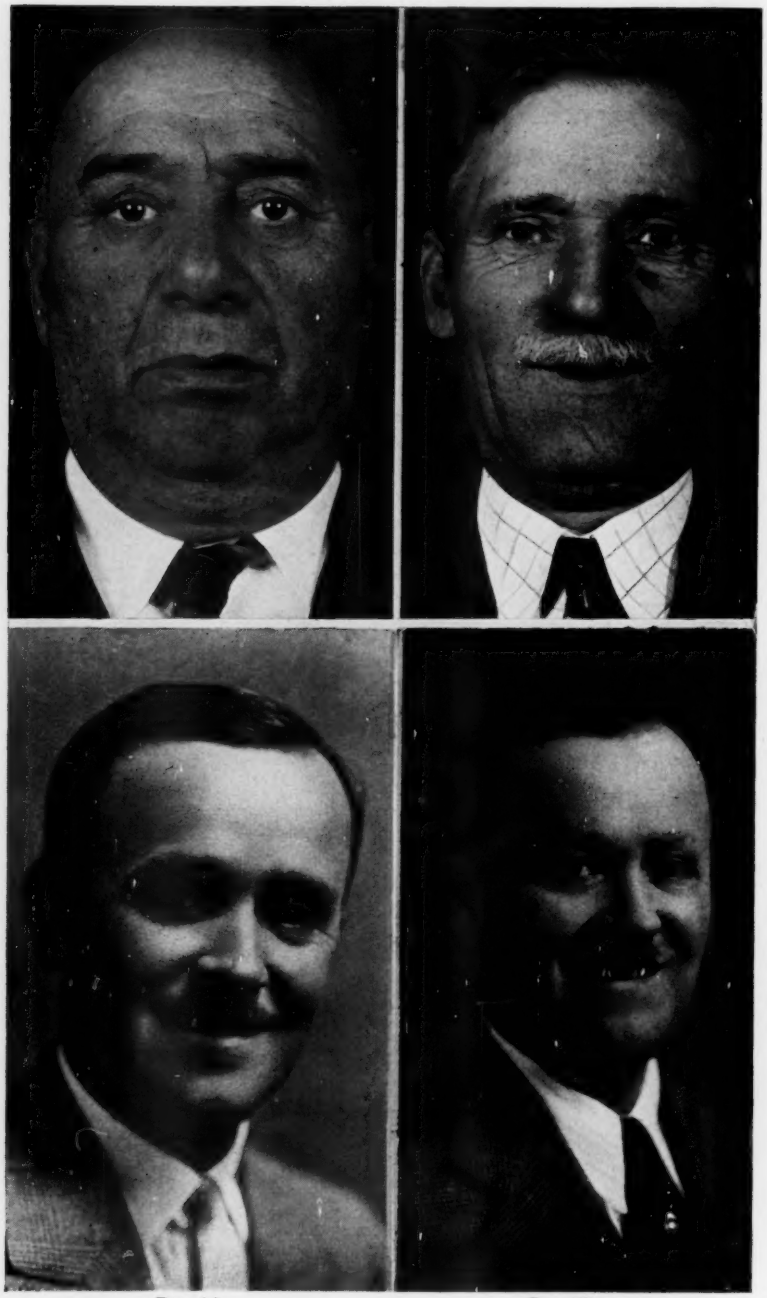


FIG. 8A. FIG. 8B.

FIG. 6.—Z. K., age 68. November, 1939. Five years and five months after operation. B.M.R. −7.

FIG. 7.—H. B., age 62. December, 1939. Five years and three months after operation. B.M.R. −16.

FIG. 8A.—L. B., age 43. 1927. Seven years before operation.

FIG. 8B.—L. B., age 53. September, 1937. Three years and six months after operation. B.M.R. −15.

FIG. 9.

FIG. 10.

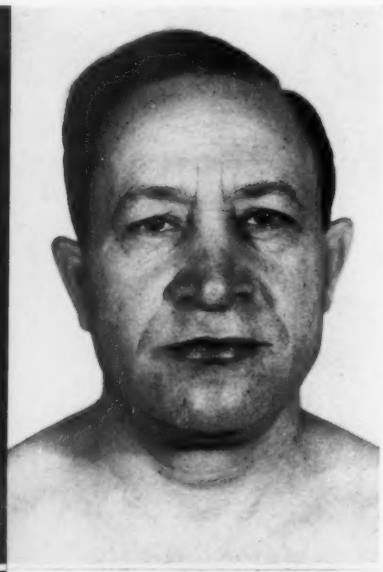


FIG. 11.

FIG. 12.

FIG. 9.—C. C., age 39. December, 1939. Six years and two months after operation. B.M.R. −21.

FIG. 10.—A. P., age 49. November, 1939. Five years and 10 months after operation. B.M.R. −28.

FIG. 11.—M. A., age 40. December, 1939. Five years and eight months after operation. B.M.R. −26.

FIG. 12.—S. B., age 35. December, 1939. Five years and seven months after operation. B.M.R. −20.

TABLE VIII

CASE SUMMARIES

ANGINA PECTORIS—FIVE-YEAR SURVIVORS—12 CASES

Case	Age and Sex	Preoperative: Duration of Symptoms	Preoperative: Coronary Thrombosis	Date of Operation	Date of Follow-Up	Postoperative Survival		Cardiovascular Status*: Symptoms	Cardiac Enlargement	Blood Pressure	Congestive Failure: Symptoms	Congestive Failure: Signs	Vital Capacity	B.M.R.	Medication: Nitroglycerin	Medication: Digitalis	Medication: Thyroid†	Myxedema	Comment
M. P.	60 F.	8 yrs.	No	8-22-33	4-20-40 Home visit	6 yrs., 7 mos.	Pre-op.	Averages 2 attacks daily, on bed and chair existence. Slight effort brings on pain.	No	175/ 70	Mild	Mild	2,200 cc.	+9	Yes	No	No	Feels cold easily. Is normal mentally but feels "slowed down."	Improved by operation for 1-2 yrs., but not now. Daughter says patient is "much better since operation but won't admit it." Has developed evidence of marked hypertension and congestive failure. A partial success.
							5 yrs. +	Bed and chair existence. Still occasional pain. Climbs 2 flights once a day	Yes	260/110	Yes	Yes	?	−15 (1935)	1-2 daily	0.1 Gm. daily	15 mg. every 2 days		
M. G.	55 M.	4 yrs.	No	9-22-33	11-27-39 Office visit	6 yrs., 2 mos.	Pre-op.	Greatly restricted activity. 1-3 attacks daily. A few steps may bring on severe pain.	No	150/ 90	Sl.	No	2,600 cc.	+7	Up to 100 a week	No	No	Mild symptoms. Feels cold easily. May have slight retardation of memory	Definitely improved by operation. Greater activity; less pain; practically no nitroglycerin. Is developing congestive failure. A good result
							5 yrs. +	Slight pain 1-2 times daily, not as severe as before operation. Climbs 2 flights twice daily slowly.	Yes	210/110	Mod.	Mod.	2,800 cc.	+5	One a month	0.1 Gm. daily	15 mg. daily		
R. H.	42 M.	21 yrs.	No	10-17-33	12-12-39 Office visit	6 yrs., 1 mo.	Pre-op.	An active clerk. Up to 20 attacks of numbness and oppression in chest daily; wide radiation. Previous bilateral cervical sympathectomy without relief.	No	160/ 90	No	No	3,500 cc.	−12	20 a day	No	No	Mild myxedema with thickened speech and subjective "poor concentration."	Improved by operation in some respects, not in others. Objectively no change. Obscure case with angina at a very early age and no bad consequences. Diagnosis may be in error. A poor result.
							5 yrs. +	Unchanged. Still at work. Still 20 attacks daily.	No	130/95	No	No	3,000 cc.	−27	20 a day	No	30 mg. daily		
J. S.	61 F.	5 yrs.	? mild attack in 1928	11- 1-33	5-17-40 Office visit	6 yrs., 6 mos.	Pre-op.	Moderately restricted activity. Attacks even at rest.	Sl.	140/ 70	No	No	3,000 cc.	−7	One or more daily	No	No	Mild symptoms. "Brain doesn't work as it should," but she worries less.	"100 per cent improved. Better than 10 years ago." Fairly active woman. Is developing evidence of hypertension and congestive failure. A good result.
							5 yrs. +	Rare attacks, usually in bed. May be free from pain for weeks. Avoids strains. Travels. Climbs 1 flight daily.	Sl.	220/110	Sl.	Mod.	2,400 cc.	−7	One a month	0.1 Gm. daily	30 mg. 5 times weekly		
G. S.	66 M.	10 yrs.	No	11-13-33	4-18-40 Phone	6 yrs., 5 mos.	Pre-op.	Attacks even at rest, requiring up to 30 nitroglycerin pills daily.	No	150/ 90	Sl.	No	2,100 cc.	−4	20-30 daily	No	No	At one time had marked myxedema. Not seen recently. Psychotic and uncooperative.	Had temporary relief for some months. Hard to appraise now, but evidently unsatisfactory. A poor result.
							5 yrs. +	Markedly psychotic. Takes many pills. Complains of severe pain.							20-30 daily	—	45 mg. daily		
W. D.	54 M	8 yrs	No	1-17-34	11-28-39 Office visit	5 yrs., 10 mos.	Pre-op.	2-3 attacks daily, occasionally at rest. Restricted to house.	No	130/ 85	No	No	3,550 cc.	−3	Yes	No	No	Feels cold easily. No mental symptoms.	Very much improved by operation. A good result.
							5 yrs. +	Mild substernal distress every 2-3 days. Less severe than before operation. Drives a car.	No	120/ 80	No	No	3,200 cc.	−11	No	No	15 mg. twice weekly		
H. Z.	56 M.	4 yrs.	1930	3- 5-34	12- 9-39 Office visit	5 yrs., 9 mos.	Pre-op.	Attacks of short duration and only upon effort. Can walk 1 block.	Sl.	145/ 85	Sl.	No	2,600 cc.	−4	Yes	Yes	No	Only slightly diminished speed of cerebration.	Feels improved by operation. Now, 5 years later, not much better than previously, but has had 4 years of sustained relief. Fatigues easily. Is developing congestive failure. A good result.
							5 yrs. +	Some recurrence of pain after 4 years of freedom. Activity restricted.	Yes	108/ 76	Yes	Yes	2,300 cc.	−15	2-8 daily	0.1 Gm. daily	15 mg. daily		
A. K.	72 M	7 mos.	No	3- 6-34	4-20-40 Home visit	6 yrs., 1 mo.	Pre-op.	Severe and prolonged pain even at rest. Not greatly relieved by nitrites.	No	160/ 85	No	No	2,300 cc.	−1	Amyl nitrite	No	No	Moderate myxedema. Slow speech, but alert mentally. Feels cold easily.	A feeble man, age 78, who was regarded as a dubious case for operation. Very emphatic about the good results of operation and permanent relief from terrific pain. Is developing congestive failure. A good result as regards pain.
							5 yrs. +	No pain since operation, although "tiredness" in arms. Bed and chair existence.	No	220/105	Yes	Yes			No	No	No		
B. F.	50 M.	3 mos.	No	4-23-34	11-28-39 Office visit	5 yrs., 7 mos.	Pre-op.	Several attacks daily even at rest. Confined to house.	No	150/ 86	Sl.	Sl.	2,400 cc.	+13	2-3 daily	No	No	Somewhat slowed mentally.	Feels greatly improved by operation. A good result.
							5 yrs. +	Rare pain, in winter only. Climbs 2-3 flights slowly. Drives	No	140/ 85	Sl.	No	2,800 cc.	−14	9 pills in 5	No	15 mg. daily		

						5 yrs. +	No pain since operation, although "tiredness" in arms. Bed and chair existence.	No	220/105	Yes	Yes				No	No	No			Feels cold easily.	The good results of operation and permanent relief from terrific pain. Is developing congestive failure. A good result as regards pain.
B. F.	50 M.	3 mos.	No	4-23-34	11-28-39 Office visit	5 yrs., 7 mos.	Pre-op.	Several attacks daily even at rest. Confined to house.	No	150/ 80	Sl.	Sl.	2,400 cc.	+13	2-3 daily	No	No	Somewhat slowed mentally.	Feels greatly improved by operation. A good result.
							5 yrs. +	Rare pain, in winter only. Climbs 2-3 flights slowly. Drives a car, but not in traffic.	No	140/ 85	Sl.	No	2,800 cc.	−14	9 pills in 5 months	No	15 mg. daily		
S. G.	50 F	3 yrs.	1931	4-27-34	11-29-39 Office visit	5 yrs., 7 mos.	Pre-op.	Spontaneous myxedema, 12 yrs. Mild angina. 1 flight produces pain.	Yes	170/ 75	Mod.	Sl.	1,600 cc.	−25	Yes	Yes	15 mg. daily	Rather marked myxedema with slow speech, *etc.*	Complicated by spontaneous myxedema. Felt improved for several years after operation, but not now. A partial success.
							5 yrs. +	Relief from pain for 1-2 yrs. Attacks now up to 4 times daily. Does light housework.	Yes	195/ 85	Mod.	Mod.	1,600 cc.	−16	Four times a day	Off and on	15 mg. daily		
Z. K.	63 M.	7 mos.	No	6-25-34	11-24-39 Office visit	5 yrs., 5 mos.	Pre-op.	Up to 10 attacks daily even at rest.	No	140/ 90	Sl.	No	—	−10	Yes	No	No	Thinks cerebration slowed.	Improved by operation. A good result.
							5 yrs. +	Very rare pain. Can walk 10 blocks. Feels quite comfortable.	No	120/ 80	Sl.	No	2,500 cc.	−7	No	0.1 Gm. daily	15 mg. daily		
H. B.	57 M.	4 yrs.	1930	8-27-34	12- 5-39 Office visit	5 yrs., 3 mos.	Pre-op.	1-2 attacks daily even at rest. Bed rest most of the day.	No	120/ 75	No	Sl.	2,900 cc.	−5	1-2 daily	No	No	No symptoms.	Very much improved. A good result.
							5 yrs. +	Rare and moderate substernal pain on effort. 1 flight twice a day. Can walk a mile.	? sl.	128/ 90	No	Sl.	3,350 cc.	−16	2-3 a year	No	15 mg. every 2 days		

* No auricular fibrillation in any case. † The figures for thyroid intake were taken from our last follow-up visit and do not represent the continuous requirement of thyroid extract which must be frequently adjusted.

TABLE IX

CASE SUMMARIES

ANGINA PECTORIS—20 DEATHS

					Preoperative Cardiovascular Status*									
Case	Age and Sex	Preoperative Duration of Symptoms	Date of Operation	Symptoms	Preoperative Coronary Thrombosis	Cardiac Enlargement	Blood Pressure	Congestive Failure: Symptoms	Congestive Failure: Signs	Cause of Death	Date of Death	Postoperative Survival	Duration of Postoperative Improvement	Comment
D. G.	43 M.	9 yrs.	12-14-32	Attacks with slightest effort, requiring up to 20 nitroglycerin tablets daily.	No	Yes	165/110	Yes	Yes	Generalized thromboangiitis obliterans. (autopsy)	9- 7-33	8 mos.	7 mos.	Free from pain for 7 months, although congestive failure was not much influenced.
A. R.	61 F.	3 yrs.	2- 4-33	3-4 attacks daily, even at rest. Bed and chair existence.	1931	Sl.	190/100	Yes	Yes	Coronary thrombosis. (autopsy)	9-18-35	2 yrs., 7 mos.	1 yr., 3 mos.	Moderately severe recurrent angina after 15 months of definite relief.
A. H.	59 M.	2 yrs.	7- 8-33	5-6 severe attacks daily, even at rest.	No	Yes	140/ 45	Yes	Yes	Acute pulmonary edema. (letter)	7-26-33	18 days	—	Only case in series with angina from syphilitic aortitis.
A. P.	58 M.	3 yrs.	9-21-33	2-4 attacks daily from slightest effort.	1931 and 1932	No	145/ 80	No	No	Old coronary sclerosis. Congestive failure. (autopsy)	4- 1-38	4 yrs., 6 mos.	3 yrs.	Recurrent angina after 3 years of relief. Coronary thrombosis twice after operation.
I. S.	64 M.	3 yrs.	9-29-33	3-4 severe attacks weekly, even at rest.	1931	No	140/ 85	Yes	Yes	Coronary thrombosis. (autopsy)	7-11-34	9 mos.	7 mos.	Complicated by mild diabetes. Definite improvement for at least 7 months, although still mild distress.
M. K.	60 F.	9 mos.	10-25-33	Frequent attacks, even at rest.	Sept. 1933	Sl.	140/ 80	Mild	Mild	Sudden death in chair. (death certificate)	8-21-37	3 yrs., 9 mos.	1 yr., 3 mos.	After 15 months of great subjective and objective relief recurrent angina and progressive congestive failure developed.
J. M.	52 F.	5 yrs.	10-26-33	Frequent attacks, even on bed rest.	1931 and Aug. 1933	Yes	140/100	Yes	Yes	"Myocarditis" and congestive failure. (death certificate)	4-25-35	1 yr., 6 mos.	1 yr., 4 mos.	Relief from pain for at least 16 months. No effect on congestive failure.
B. A.	61 M.	2 yrs.	10-28-33	Frequent attacks, with slightest effort.	No	Yes	120/ 90	No	No	Pulmonary edema. (coroner)	1-12-38	4 yrs., 2 mos.	2 yrs., 10 mos.	Definite relief for 2 years 10 months. Some recurrent angina after 4 years.
O. H.	68 M.	2 yrs.	10-28-33	Severe attacks, even with the slightest effort.	? Mild attack, 4 months before operation.	No	145/ 90	No	No	Chronic myocarditis. (death certificate)	10- 5-36	2 yrs., 11 mos.	1 yr., 6 mos.	Complicated by mild diabetes. Definite relief for at least 18 months, although recurrent, but atypical, angina 2½ years later.
K. W.	65 F.	12 yrs.	11- 2-33	Up to 15 attacks daily, even at rest.	No	Sl.	190/ 90	No	No	Coronary thrombosis. (death certificate)	2-17-35	1 yr., 3 mos.	6 mos.	Considerable improvement for at least 6 months, although continued nitroglycerin. Recurrent angina after 1 year.

TABLE IX (Continued)

CASE SUMMARIES
ANGINA PECTORIS—20 DEATHS

Case	Age and Sex	Preoperative Duration of Symptoms	Date of Operation	Symptoms	Preoperative Cardiovascular Status*				Cause of Death	Date of Death	Postoperative Survival	Duration of Postoperative Improvement	Comment
					Preoperative Coronary Thrombosis	Cardiac Enlargement	Blood Pressure	Congestive Failure: Symptoms	Congestive Failure: Signs					
W. S.	67 M.	10 mos.	11-22-33	Frequent and severe attacks, even at rest.	No	No	140/ 80	No	No	Coronary thrombosis. (autopsy)	11-27-33	5 days	—	Postoperative death.
I. C.	65 F.	8 mos.	11-24-33	Up to 20 severe attacks daily, even at rest.	? mild attack, 8 months before operation.	No	160/ 80	No	No	Congestive failure. (death certificate)	10-25-37	3 yrs., 10 mos.	3 yrs., 5 mos.	Complete relief from pain for at least 3 years, 5 months.
C. W.	53 M.	5 yrs.	12- 7-33	Attacks requiring up to 10 nitroglycerin tablets daily, even at rest.	1932	No	110/ 70	No	No	Coronary thrombosis. (autopsy)	4- 2-34	3 mos.	1 mo.	Recurrent angina after complete relief for 1 month.
N. N.	61 M.	12 yrs.	12- 8-33	Many attacks daily, even at rest.	? attack in 1927	No	220/110	Yes	No	Cerebral hemorrhage. (death certificate)	8- 9-38	4 yrs., 8 mos.	2 yrs.	Intermittent fibrillation. Symptoms from tetany. Recurrent angina after 2 years of definite improvement.
W. R.	65 M.	1 yr.	12-13-33	1-4 attacks daily, usually at night.	No	No	120/ 80	No	No	Congestive failure. (death certificate)	2-16-36	2 yrs., 8 mos.	1 yr., 2 mos.	Occasional fleeting pains, but definite relief for at least 14 months. Some symptoms from myxedema.
E. L.	67 F.	7 yrs.	12-13-33	4-10 attacks daily, even at rest.	—	Sl.	140/ 70	No	No	Coronary thrombosis. (autopsy)	12-14-33	1 day	—	Postoperative death.
S. S.	62 F.	1 yr.	1- 4-34	4-6 attacks daily, even at rest.	No	Sl.	165/ 95	Sl.	Yes	Pulmonary edema. (hospital—no autopsy)	1- 5-34	1 day	—	Complicated by mild diabetes. Postoperative death.
L. S.	64 F.	3 yrs.	1-20-34	Frequent attacks. Bed rest for 2 yrs.	No	No	220/130	? no	No	Coronary thrombosis. (letter)	2- 5-35	1 yr.	9 mos.	Definite relief from pain, but marked discomfort from myxedema.
L. B.	50 M.	4 yrs.	3-21-34	3 attacks daily.	4 months before operation.	No	110/ 65	Yes	No	Coronary thrombosis. (autopsy)	1- 2-38	3 yrs., 9 mos.	3 yrs., 6 mos.	Marked and sustained relief, practically to time of death.
E. S.	65 F.	3 yrs.	4-12-34	Constant precordial pressure and frequent sharp attacks.	No	Sl.	130/ 80	Yes	No	"Myocarditis." (death certificate)	1-13-37	2 yrs., 9 mos.	3 mos.	Complicated by recurrent hyperthyroidism. Subtotal thyroidectomy in 1931, with relief for 2 years. Following total thyroidectomy no definite relief after 3 months. Definite symptoms by 11 months.

* Only one case had auricular fibrillation (N. N.).

TABLE X

CASE SUMMARIES
CONGESTIVE FAILURE IN CHRONIC RHEUMATIC VALVULAR DISEASE—16 CASES

Case	Age and Sex	Preoperative Duration of Symptoms	Date of Operation	Symptoms	Preoperative Cardiovascular Status†				Cause of Death	Date of Death	Postoperative Survival	Duration of Postoperative Improvement	Comment
					Cardiac Diagnosis*	Auricular Fibrillation	Blood Pressure	Vital Capacity‡					
B. M.	41 F.	23 yrs.	4- 7-33	Recurrent decompensation.	MS & MI. Calcified pericardium	Yes	145-110	1,100 cc.	Congestive failure. (autopsy)	10-17-35	2 yrs., 6 mos.	7 mos.	Definitely, although temporarily, relieved. Frequent postoperative hospitalization for decompensation.
F. R.	53 M.	9 yrs.	5-19-33	Recurrent decompensation.	MS & MI	Yes	210/ 68	1,650 cc.	Congestive failure. (autopsy)	10- 8-33	4 mos.	—	
S. P.	45 M.	2 yrs.	5-26-33	Recurrent ascites (10 paracenteses).	MS & MI. Adherent pericardium	Yes	120/ 70	2,100 cc.	Postoperative death, after pericardiectomy. (hospital—no autopsy)	7-22-33	1 mo.	—	Pericardiectomy in this hospital.
D. W.	28 F.	8 yrs.	6- 5-33	Recurrent decompensation.	MS & MI	No	170/ 85	1,800 cc.	Congestive failure. (letter from physician)	3-21-34	9 mos.	6 mos.	Questionably improved for 6 months, then progressive failure. Markedly psychotic after operation.
C. C.	33 M.	5 mos.	10-16-33	Progressive severe decompensation.	MS & MI	Yes	140/ 90	2,000 cc.	Congestive failure. (hospital—no autopsy) 5-year survival.	6-21-40	6 yrs., 8 mos.	6 yrs.+	Improved subjectively and objectively, although numerous hospital admissions for mild decompensation.
E. F.	54 M.	12 yrs.	12- 6-33	Severe dyspnea without right ventricular failure.	MS & MI	Yes	140/ 80	2,000 cc.	Congestive failure. (hospital—no autopsy)	12- 1-38	4 yrs., 11 mos.	4 yrs.+	Excellent result until terminal illness.
P. R.	39 F.	11 yrs.	1-27-34	Recurrent decompensation.	MS & MI	Yes	125/ 90	2,100 cc.	Congestive failure. (autopsy)	11-29-38	4 yrs., 10 mos.	2 yrs.+	Moderate improvement for at least 2 years.
F. M.	50 M.	10 yrs.	2- 8-34	Recurrent decompensation.	MS	Yes	140/ 80	1,200 cc.	Congestive failure. (autopsy)	4-21-35	1 yr., 2 mos.	2 mos.	Progressive failure after short improvement.
A. P.	44	7 yrs.	2-12-34	Progressive decompensation.	MS	Yes	140/ 80	2,200 cc.	Living Dec., 1939. 5-year survival	—	5 yrs., 10 mos.	5 yrs.+	Improved subjectively and objectively. Can climb 2 flights. Vital capacity 2,100 cc. B.M.R.—28 on

E. P.	34 M.	12 yrs.	12- 6-33	Severe dyspnea without right ventricular failure.	MS & MI	Yes	140/ 80	2,000 cc.	Congestive failure. (hospital —no autopsy)	12- 1-38	4 yrs., 11 mos.	4 yrs.+	Excellent result until terminal illness.
P. R.	39 F.	11 yrs.	1-27-34	Recurrent decompensation.	MS & MI	Yes	125/ 90	2,100 cc.	Congestive failure. (autopsy)	11-29-38	4 yrs., 10 mos.	2 yrs.+	Moderate improvement for at least 2 years.
F. M.	50 M.	10 yrs.	2- 8-34	Recurrent decompensation.	MS	Yes	140/ 80	1,200 cc.	Congestive failure. (autopsy)	4-21-35	1 yr., 2 mos.	2 mos.	Progressive failure after short improvement.
A. P.	44 M	7 yrs.	2-12-34	Progressive decompensation.	MS	Yes	140/ 80	2,200 cc.	Living Dec., 1939. 5-year survival.	—	5 yrs., 10 mos.	5 yrs.+	Improved subjectively and objectively. Can climb 2 flights. Vital capacity 2,100 cc. B.M.R.—28 on 15 mg. thyroid a day. Moderate myxedema.
S. C.	30 F.	8 yrs.	2-13-34	Recurrent decompensation.	MS, MI, AS & AI	No	110/ 70	1,700 cc.	Congestive failure. (hospital —no autopsy)	2- 7-37	2 yrs., 11 mos.	2 yrs.+	Fairly well for over 2 years. Then gradual failure.
L. A.	24 M.	22 yrs.	4-23-34	Recurrent decompensation.	MS, MI & AI	Yes	140/ 60	1,350 cc.	Congestive failure. (death certificate)	8-26-34	4 mos.	—	Unimproved. A cardiac from the age of 2.
M. A.	35 F.	17 yrs.	4-25-34	Recurrent severe decompensasation—6 months. Milder symptoms for a long time.	MS & MI	Yes	150/ 90	2,100 cc.	Living Dec., 1939. 5-year survival.	—	5 yrs., 8 mos.	2 yrs.+	Markedly improved for 2 years. Can walk 1 block. Can climb 1 flight. Vital capacity 1,600 cc. B.M.R. —26 on 15 mg. thyroid a day. No myxedema.
S. B.	30 M.	15 yrs.	5-26-34	Recurrent decompensation.	MS & AI	No	130/ 90	1,800 cc.	Congestive failure and pulmonary embolism. (hospital —no autopsy) 5-year survival.	2-27-40	5 yrs., 9 mos.	5 yrs.+	Definitely improved until terminal illness. Worked intermittently. Climbed 1 flight.
H. B.	46 F.	6 yrs.	6- 2-34	Recurrent decompensation. Bed rest for 4 months.	MS & MI	Yes	204/140	1,200 cc.	Sudden death. ? pulmonary embolism. (hospital—no autopsy)	6- 3-34	1 day	—	Postoperative death.
M. B.	50 M.	6 mos.	8- 1-34	Progressive decompensation.	MS	Yes	186/134	1,100 cc.	Slow coronary closure. (letter from physician)	4-23-37	2 yrs., 8 mos.	2 yrs.+	Some improvement for 2 years.
E. P.	31 F.	13 yrs.	12-12-34	Recurrent decompensation.	MS, MI, AS & AI	No	150/ 60	2,400 cc.	Cerebral emboli. (hospital— no autopsy)	2- 8-35	1 mo.	—	Decompensation improved after operation.

* MS—mitral stenosis. MI—mitral insufficiency. AS—aortic stenosis. AI—aortic insufficiency.
† Cardiac enlargement and evidence of congestive failure in all 16 cases.
‡ The vital capacity reading is the one just before operation, and may have been lower earlier.

TABLE XI

CASE SUMMARIES

CONGESTIVE FAILURE IN ARTERIOSCLEROTIC OR HYPERTENSIVE HEART DISEASE—9 CASES

					Preoperative Cardiovascular Status*								
Case	Age and Sex	Preoperative Duration of Symptoms	Date of Operation	Symptoms	Cardiac Diagnosis	Auricular Fibrillation	Blood Pressure	Vital Capacity†	Cause of Death	Date of Death	Postoperative Survival	Duration of Postoperative Improvement	Comment
R. M.	61 M.	4 yrs.	3- 9-33	Severe decompensation.	Chronic myocarditis	Yes	136/ 90	1,950 cc.	Chronic myocarditis heart block. (hospital—no autopsy)	8-27-33	5 mos.	3 mos.	
G. C.	70 M.	13 yrs.	4-11-33	Coronary thrombosis, with progressive decompensation—2½ weeks.	Chronic myocarditis. Coronary sclerosis	No	156/ 88	1,200 cc.	Coronary thrombosis and congestive failure. (autopsy)	4-11-33	..	—	Postoperative death. A desperate case.
L. W.	48 M.	4 yrs.	8- 3-33	Recurrent decompensation. Some chest pain.	Chronic myocarditis	Yes	190/ 96	2,250 cc.	Lobar pneumonia. No cardiac failure. (autopsy)	June, 1937	3 yrs., 10 mos.	3 yrs.+	Improved until death from another cause. Best result in this group.
H. E.	40 M.	5 yrs.	1-13-34	Severe decompensation. Bed rest for 4 months.	Chronic myocarditis	No	120/ 80	1,700 cc.	Pulmonary edema. (hospital letter)	2-16-34	1 mo.	—	
P. S.	50 F.	4 yrs.	2- 1-34	Recurrent decompensation.	Chronic myocarditis	No	174/110	1,750 cc.	"Chronic interstitial nephritis." Chronic myocarditis. (death certificate)	5-23-35	1 yr., 3 mos.	2 mos.	Slight improvement. Only follow-up contact 2 months after operation.
O. McG.	50 M.	5 yrs.	6-25-34	Severe dyspnea. Bed rest for 6 weeks.	Hypertensive cardiovascular disease	No	170/120	1,600 cc.	"Arteriosclerosis. Chronic nephritis and uremia." (death certificate)	1-21-36	1 yr., 7 mos.	6 mos.	Temporary relief in a previously bed-ridden man.
H. W.	56 M.	3 yrs.	7-19-34	Progressive decompensation.	Chronic myocarditis. Coronary disease	No	125/ 70	2,000 cc.	Congestive failure. (letter from physician)	10-15-34	2 mos.	—	Unimproved. Not much cardiac enlargement.
M. K.	37 M.	6 mos.	8- 9-34	Progressive decompensation.	Hypertensive vascular disease. Nephrosclerosis	No	240/150	—	Bronchial pneumonia. No cardiac failure. (letter from physician)	9-23-34	1 mo.	—	
S. L.	36 M.	6 mos.	8-15-34	Dyspnea and constant precordial tightness.	Hypertensive cardiovascular disease	No	210/ 80	1,200 cc.	Congestive failure. (letter from wife)	2-28-39	4 yrs., 6 mos.	2 yrs., 6 mos.	Definite improvement for 2½ years, although somewhat troubled by myxedema. Last follow-up April, 1937.

* Cardiac enlargement (slight only in H. W.) and evidence of congestive failure in all cases.
† The vital capacity reading is the one just before operation, and may have been lower earlier.

and 1934. The majority of the patients had been unrelieved by medical therapy and presented a serious operative risk. There were 12 survivors in the group of 32 with angina pectoris, and four survivors in the group of 25 who had congestive failure. There were five postoperative deaths; four of these, as well as all but three of the later deaths, were attributable to heart disease.

The best results were obtained in patients with angina pectoris. Twenty-six of the 27 patients surviving more than six months were relieved of pain in some degree for six months or longer, and eight of the 12 five-year survivors had sustained relief. In this group it was noted that preoperative evidence of congestive failure or cardiac enlargement was an unfavorable prognostic sign for long survival.

In the patients with congestive failure the five-year results were disappointing. Fifteen of the 25 patients lived for six months or more, and 12 of these had relief for six months or longer. There were four five-year survivors, three showing sustained relief; two of these three have died of congestive failure in the sixth year after operation. Results were better in the group having congestive failure from chronic rheumatic valvular disease than from arteriosclerotic or hypertensive heart disease.

We conclude that in a selected group of patients with intractable angina pectoris, total thyroidectomy is a worth while therapeutic measure, and is without unwarranted risk.

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CHRONIC GASTRIC ULCER, IN CHILDHOOD, TREATED SURGICALLY

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CHRONIC GASTRIC ULCER is a comparatively rare disease in children. Proctor[1] from this clinic reported one instance of chronic gastric ulcer in a child encountered in a series of approximately 1,600 cases of chronic gastric ulcer during the years 1906 to 1924. He collected 11 other cases from the literature. Foshee,[2] in 1932, found six other reported cases and added one of his own, making a total of 19 cases. Since that time, 15 other case reports have been found in the available literature.* All of these children were 14 years of age or younger. They had had symptoms for at least two months or their ulcers showed pathologic evidence of chronicity (Case 15, Colson, Table I). These criteria are similar to those of Proctor[1] and Foshee.[2] Pathologic reports were not available in some cases as operative treatment had not been necessary in all instances. The acute ulcer of the neonatal and early period of infancy is excluded from consideration here.

At the Mayo Clinic, from 1924 to 1939, inclusive, there have been seen approximately 2,000 cases of chronic gastric ulcer, in only one of which was the subject a child. Duodenal ulcer has been observed more frequently among children than has gastric ulcer. This is in keeping with the general frequency of the two types of ulcer.[4]

The etiology of gastric ulcer in childhood remains the same unsolved and much discussed problem as that which occurs in adult life. In this article we will refrain from entering the controversy.

The diagnosis is often difficult owing, chiefly, to the rarity of gastric ulcer in this age-group and to the bizarre symptoms which it produces in children. "The most important single factor in the diagnosis is the realization that chronic peptic ulcer occurs in children."[1] Chronic digestive disturbances, indefinite abdominal discomfort, especially epigastric in situation, anorexia, night pain and vomiting should suggest the possibility of peptic ulcer. Chronic constipation of varying degrees often occurs. The back pain in the case which we are reporting was difficult to account for. This is generally associated with a perforating ulcer into the pancreas or into the gastrohepatic omentum,[5]

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* Rocher,[3] in 1934, quoting Depiersis, stated that 53 cases of gastric ulcer in childhood had been reported up until that time but he made no distinction between acute and chronic ulcers and unfortunately published no references.

TABLE I

CASES OF CHRONIC GASTRIC ULCER AMONG CHILDREN RECORDED IN THE AVAILABLE LITERATURE SINCE THE REPORT OF FOSHEE,[2] 1932

Author	Reference	Age and Sex	Duration of Symptoms	Site of Lesion	Method of Diagnosis	Therapy
Robinson, V. P.	Lancet, **2**, 600, September 17, 1927	12 yrs. M.	3 mos.	Anterior wall of stomach, halfway between fundus and pylorus; nearer lesser than greater curvature (ruptured).	Surg.	Surg.
Blechmann, G., Gutmann, R. A., and Nemours-Auguste	Nourrisson, **20**, 34–37, 1932	10 mos. M.	2 mos.	Pylorus.	Roent.	Med.
Oldfield, M.	Brit. Med. Jour., **1**, 836–837, May 7, 1932	13 yrs. M.	2 yrs.	Posterior surface close to lesser curvature, 3 in. from pylorus.	Roent.	Med.
Jankelson, I. R.	Am. Jour. Dis. Child., **44**, 162–165, July, 1932	12 yrs. M.	5 to 6 yrs.	Lesser curvature.	Roent.	Med.
		12 yrs. F.	7 mos.	Prepyloric.	Roent.	Med.
Olper, Leone	Arch. ital. d. mal. d. app. diger., **2**, 39–51, 1933. Abst. Zentralbl. f. d. ges. Kinderh., **28**, 335, 1933–1934	11 yrs. M.	Not given	Pyloric bulb.	Roent. and surg.	Surg.
Sáinz, de les Terreros, C., and Pérez Moreno, B.	An. Hosp. de San Jose y Santa Adela., **5**, 285–294, 1933–1934; and Arch. espân. de pediat., **18**, 522–534, September, 1934.	5 mos. M.	4 mos.	Lesser curvature near antrum.	Roent.	Med.
Rocher, H. L.[3]	Rev. franç de pediat., **10**, 218–224, 1934	8 yrs. M.	4 yrs.	Anterior superior wall of pylorus.	Roent. and surg.	Surg.
Micheli, E.	Boll. e. mem. Soc. piemontese di chir., **4**, 467–480, 1934	13 yrs. F.	1 yr.	Pylorus.	Roent. and surg.	Surg.
		12 yrs. M.	4 yrs.	Pylorus.		
Toro, N.	Pediatria, **45**, 904–923, October, 1937	10 yrs. F.	1½ yrs.	Pylorus.	Clin., surg. and roent.	Surg.
		11 yrs. M.	1 yr.	Pylorus.		Surg.
Bertrand, J. C., Messina, Bernardo, and de la Fare, Mauricio	Arch. argent. de pediat., **8**, 990–996, 1937. Abst. Zentralbl. f. d. ges. Kinderh., **34**, 348, 1937–1938	12 yrs. F.	2 yrs	Pylorus.	Roent. and surg.	Surg.
Webster, Reginald	Med. Jour. Austral., **1**, 1061–1062, 1938	4 mos. M.	3 mos.	Posterior wall at pylorus.	Necropsy*	
Colson, Cade, R., and Soustelle	Lyon. méd., **162**, 35–37, July 10, 1938	14 yrs. M.	Few hrs.†	Prepyloric (ruptured).	Surg.	Surg.

* Recovery occurred in all cases except that reported by Webster.
† Ulcer indurated at operation.

but in our case there was no perforation. Low abdominal pain as observed in the case reported is another bizarre symptom for which we can offer no explanation. Hematemesis, melena or both are very suggestive of peptic ulcer. The roentgenologic picture is characteristic.[6] Whereas the latter statement is generally true, Eusterman and Balfour[4] stated that a competent roentgenologist can demonstrate 96 per cent of gastric ulcers, Kennedy[7] and Armingeat[8] have reported instances in which the surgeon could not substantiate the roentgenologist's report of ulcer but in which he found appendiceal inflammation. However, in both of these cases, the stomach and duodenum were examined without being opened. Gastroscopic examination should be carried out in doubtful cases.

It is generally agreed that the treatment for gastric ulcer is medical, except in cases in which there is perforation, suspicion of malignancy or failure of a medical regimen. In these latter cases, surgical treatment is indicated. Since the medical regimen is essentially the same for children as for adults,[9, 10] it will not be discussed here.

Case Report.—A white girl, age 12, was referred to the clinic, March 21, 1939, because of abdominal pain, vomiting and melena. The mother was considered "nervous" and was moderately deaf. The family history otherwise was not remarkable. The patient had always been considered to be a "nervous child." She had had measles, mumps, pertussis and chickenpox. She had had a tonsillectomy and adenoidectomy performed, two years previously.

During the year preceding admission, the patient had had some intermittent backache and abdominal pain. The abdominal discomfort apparently had been worse during the few months preceding registration at the clinic, and the backache, during the three weeks preceding admission. During the week previous to examination, the abdominal pain had been more severe and cramp-like, and was often worse in the lower part of the abdomen. No relation of pain to eating or to bowel movements could be determined. She had been given a cathartic once without its affecting the symptoms. During the two days before admission, she took very little orally, and had vomited dark brown to black material on three occasions. Her stools were black. At no time was there gross blood in either the stools or the vomitus.

Physical Examination.—The patient was a tall, thin, undernourished girl, with comparatively dry skin and mucous membranes. She was in no distress. The oral temperature was 99.8° F. (37.7° C.) and the pulse rate was 80. Blood pressure 130/78. The contour of the abdomen was flat. There was no spasm, mass or tenderness. Rectal examination was essentially normal, as was the rest of the examination.

The urine was normal. The concentration of hemoglobin was 12.6 Gm. per 100 cc.; erythrocytes 4,410,000; leukocytes 6,700, 27 per cent lymphocytes, 17 per cent monocytes and 56 per cent neutrophilic leukocytes. The flocculation test for syphilis and the tuberculin tests, which were made with first and second strengths of purified protein derivative, gave negative results. Blood urea 34 mg. per 100 cc. Both the benzedine and the guaiac tests for occult blood in the stool were positive. Roentgenograms of the thorax and of the dorsolumbar spine were normal. Fluoroscopic and roentgenologic examination of the stomach revealed what was interpreted as an ulcer on the lesser curvature. On reexamination, it was determined that there was an ulcer, 1.5 cm. in diameter, situated just below the angle. Gastroscopic examination revealed a lesion on the lesser curvature of the stomach which appeared to be of an infiltrating type, and, hence, one which might be malignant.

Because of the chronic, hemorrhagic nature of the lesion, and the possibility that the

lesion was malignant, an exploratory operation was decided upon, and was performed March 31, 1939 (W. W.).

Operation.—The stomach and duodenum were exposed through a midline incision. There was an ulcer situated on the lesser curvature in the region of the incisura with a crater, about 1.5 cm. in diameter, surrounded by regions of inflammation and induration of about equal size. A segmental excision of a portion of the stomach including the ulcer was made with the electrocautery, going well beyond the regions of inflammation and induration. The opening in the stomach was closed with chromic catgut and silk and was protected with omentum. The pyloric sphincter was divided longitudinally down to the mucous membrane, retracting the peritoneum and pyloric muscle laterally and covering the area with a portion of the gastrocolic omentum. At the conclusion of the operation, the appendix, which showed chronic inflammation with fibrosis, was removed. The pathologic examination of the portion of stomach removed showed a subacute hemorrhagic gastric ulcer, 2x1x5 Mm. in size. The appendix showed chronic inflammatory changes with submucous fibrosis.

The patient's postoperative course was uneventful. She was given the postoperative diet and treatment that is usual for gastro-enterostomy.[11] On the eighth day after operation, the concentration of hemoglobin was 9.7 Gm. per 100 cc. Erythrocytes 3,580,000; leukocytes 6,000. Ferrous sulfate 9 gr. (0.6 Gm.) per day was administered. The patient was discharged on the twenty-first day after operation.

The patient was reexamined four months after operation. She complained only of occasional slight abdominal pain which appeared if she ate excessive amounts of food. She had gained 3.3 lbs. (1.5 Kg.) and otherwise had remained well. Six months after operation, she was feeling very well and had had very little abdominal discomfort. She had, however, lost 4 lbs. (1.8 Kg.) since her previous visit. A program for rest was outlined. One month later, she was seen again, at which time she had regained her lost weight, but her weight was still, approximately, 25 per cent below normal. She had no complaints. Continuation of the program of rest was advised.

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HYDROCELE OF THE FEMORAL HERNIAL SAC

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An examination of the medical literature available indicates that hydrocele of the femoral canal is an uncommon condition. Only five authentic cases were found, although a few additional reports might belong in this classification.

Probably the earliest reference to the condition was that made by Sir Astley Cooper,[3] in 1844, when he wrote: "Dr. Monro, jun., mentions an instance of an hydatid tumor which was removed from the upper and inner part of the thigh, which might easily be mistaken for hernia; and he gives another example of it from Desault, who found it transparent when a candle was brought near to it, and that he could draw it from the crural arch so as to leave a space between the tumor and the abdomen, which proved that it was not formed from it." These two, however, can hardly be accepted as authentic cases of hydrocele of the femoral hernial sac.

Erdman,[5] without reporting any cases of this type, mentioned that "a small tab of inflamed omentum with serous exudate simulating hydrocele" is "frequently" present in femoral hernial sacs; and Babcock[1] wrote: "Cysts of the femoral canal are rare, irreducible and without impulse. They may be difficult to diagnose from an irreducible hernia without exploratory incision." These authors may have recognized the condition under discussion.

In 1892, the first proved case noted in the literature was reported by Marcy,[6] as follows: "Cystic dilatation of a portion of the hernial sac may form a complication. I have very recently operated upon a woman of about 40 years of age who had suffered from femoral hernia for 12 or 13 years, much of the time wearing a truss. A portion of the tumor became irreducible, and the truss could not be tolerated. The sac was thick-walled, and the lower portion was occluded and filled with serum, making a tumor the size of an egg."

DeGarmo,[4] in 1907, commented that the neck of a femoral hernial sac is narrow, becomes tough and thickened, and, if a truss has been worn, may be closed off completely. "A sac that has been closed off in this manner is quite likely to take on a condition of hydrocele, and, as it occupies the exact site of the former hernia, is very likely to be mistaken for irreducible femoral hernia." He illustrated such a case by a drawing made from a patient. The "cyst"

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was in the distal end of the sac and the "hernia proper was in the upper part of the sac and easily reduced."

In 1927, Bailey[2] reported a case of "Hydrocele of a Femoral Hernial Sac:" A woman, age 58, who had a femoral hernia of 14 years' duration, developed ascites and cardiac decompensation. After several months of treatment, the ascites disappeared, but the sac remained filled with fluid. It was aspirated and the contents subsequently became infected, requiring incision and drainage. The wound healed in two weeks and "the patient left the hospital with no sign of the femoral hydrocele."

In 1934, Rives[7] reported two cases. The first was that of a colored woman, age 33, with an asymptomatic tumor in the right groin. It had gradually enlarged to the size of a "hen's egg" during the previous five years. At operation, a thin-walled cyst containing translucent fluid was found. It communicated with the peritoneal cavity through the femoral ring. His second case was that of a colored woman, age 43, who had a mass in the left groin, of three years' duration, that had also gradually enlarged to the size of a "hen's egg." Some slight pain was associated with it at first but it later became painless. At operation, a thin-walled cyst, which communicated with the peritoneal cavity through the femoral canal, was found.

Three proved cases of hydrocele of a femoral hernial sac were seen recently on the Surgical Service of the University of California Hospital.

CASE REPORTS

Case 1.—Mrs. M. E. S., age 49, entered the University of California Hospital, April 3, 1938. She had had a small, firm lump in her left groin for about ten months. It was associated with a dragging pain which was relieved by reclining. In the left femoral region was a small, firm, tender, irreducible mass, 2.0×1.5×1.0 cm. in size. A diagnosis of femoral hernia containing incarcerated omentum was made before operation.

Operation.—April 4, 1938: Under nitrous oxide anesthesia, a left inguinal incision was made. Immediately below the inguinal ligament a rounded, fatty mass, about two centimeters in diameter, was found. Further dissection showed it to be a small hydrocele covered by a thin layer of fat. It was opened and found to contain thick yellowish fluid. The sac was lined with a smooth shining membrane. The neck of the hydrocele was dissected through the femoral canal, beneath the inguinal ligament, to its attachment to the parietal peritoneum. It was not possible to demonstrate a communication between the lumen of the sac and the peritoneal cavity. The attachment was divided and transfixed, and the femoral canal obliterated with sutures. The patient's postoperative course was uneventful and she was discharged on the eleventh day after operation. The pathologist reported that the specimen of the wall of the hydrocele was a "mesothelial-lined sac."

Case 2.—Mrs. M. C. H., age 48, entered the University of California Hospital, May 17, 1938. She complained of swelling in the right groin of 15 years' duration. At first it was about 3×2 cm. in size, but had increased gradually until about a year before her entry to the hospital, when it began to enlarge rapidly. For a short time, working or walking had caused it to become tender and painful. Examination showed a superficial mass, about 10×6×2 cm. in size, in the right groin. It was soft, rounded, fluctuant, freely movable, not tender, and seemed to be loosely attached to the deeper structures. It did not vary in size with coughing or straining. The diagnosis was somewhat uncertain, but a lipoma was thought most likely and an incarcerated hernia was considered possible.

Operation.—May 17, 1938: Under local anesthesia, an incision three inches long was made directly over the mass, which presented as a bluish, lobulated cyst. The hydrocele was dissected to the neck that entered the femoral canal. It was opened and found to contain yellow fluid, similar to that seen in hydrocele in the male. The fluid was evacuated and the neck dissected to its communication with the peritoneal cavity where it was divided and ligated. The femoral canal was obliterated with sutures. The patient left the hospital on the tenth postoperative day after an uneventful convalescence.

Case 3.—Mrs. M. M. R., age 37, entered the University of California Hospital, April 17, 1939. She complained of a swelling in the right groin of nine months' duration. During the previous four months it had increased in size and had been sharply painful at times. There had been occasional nausea but no vomiting. A small, firm mass, about two centimeters in diameter, was seen on the right side of the junction of the lower and middle thirds of the inguinal ligament. It was slightly tender and gave a slight impulse on coughing. It could not be reduced.

Operation.—April 18, 1939: Under local anesthesia, an incision was made above the right inguinal ligament. Just below the inguinal ligament there was a slightly irregular cystic mass about two centimeters in diameter, which was attached by a narrow neck through the femoral canal to the peritoneum. The external oblique muscle was opened, the femoral canal was exposed from above, and the mass was reduced through the femoral ring by traction from above. The cyst was opened and found to contain a small amount of straw-colored fluid. The lining was smooth and shining and communicated with the peritoneal cavity through a long narrow neck. The sac was dissected free of areolar tissue and closed with a suture at the neck. The hernia was repaired from above Poupart's ligament in the usual manner. The patient was discharged from the hospital on the fifteenth day after operation, with the wound healed.

Discussion.—To explain the occurrence of femoral hydrocele, it seems necessary to suppose the presence of a potential or actual femoral hernial sac or an embryologic peritoneal rest. An explanation of the appearance of the fluid is not easy. As Rives said—"the fluid rather than the hernia needs explanation, and no explanation is entirely satisfactory." He seemed to feel that the fluid from the peritoneal cavity might have gravitated to the preformed pouch and have been trapped there by adhesions across its narrow neck. Such a mechanism must certainly have produced the hydrocele in Bailey's case, in which the patient was known to have had a femoral hernial sac and to have had ascites. The additional factor of trauma, such as that caused by a truss in Marcy's and DeGarmo's patients, was not present, so far as is known, in our cases or those of Rives. Erdman suggested the factor of "inflammation." The possibility that the occlusion or isolation of the sac might in some cases be congenital is suggested by our cases, particularly the first. The accumulation of fluid in such an isolated sac would appear to be similar to that which occurs in the hydroceles of the cord and scrotum in the male and of the canal of Nuck in the female.

The diagnosis of hydrocele of the femoral hernial sac is not likely to be made before operation because of the rather frequent occurrence of incarcerated omentum in femoral hernia. Recognition at operation requires no particular consideration, however, except perhaps to note that the hydrocele should be approached from below the inguinal ligament.

SUMMARY.—Eight cases of femoral hydrocele are reported, five from the

available literature and three from our clinic. Two possible cases noted by Sir Astley Cooper, and references to somewhat similar conditions in other writings are also mentioned.

The factors involved in the formation of femoral hydrocele are discussed briefly without definite conclusion, but with the feeling that, in the authors' cases at least, congenital alterations were most likely present.

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EPIDERMOID CARCINOMA OF THE EXTREMITIES WITH REFERENCE TO LYMPH NODE INVOLVEMENT

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THE TREATMENT of epidermoid carcinoma of the extremities inevitably involves a decision in regard to the treatment of regional lymph nodes. This decision must be based upon the presence or probability of development of metastatic involvement. It is the purpose of the present study to determine what characteristics of the primary lesion are associated with the probability of metastases. The accuracy of clinical appraisal of node involvement, and the efficacy of the methods of treatment of metastases when they are present, have also been investigated.

This analysis is based upon the cases of epidermoid carcinoma observed at the Massachusetts General Hospital during the years 1922–1937, at the Huntington Memorial Hospital during the years 1915–1937, and at the Pondville Hospital during the years 1927–1937. The study is not primarily an end-result study. Untraced cases have been omitted as inconclusive. In general, cures have been followed for at least three years after the last treatment was given.

Etiology.—The location, age, and sex incidence, and delay from onset to treatment are shown in Table I.

TABLE I

AGE AND SEX INCIDENCE

Location	Males	Females	Youngest	Oldest	Median Age	Average Delay to Treatment	No. of Cases
Finger and hand....	206	78	29	95	67	1.7 years	284
Arm.....	21	20	31	92	65	1.7 years	41
Foot.....	23	9	13	79	55	1.8 years	32
Leg.....	35	38	21	80	57	2.8 years	73
Totals.....	285	145					430

It should be noted that males predominate over females in carcinoma of the finger, hand, and foot. There is no significant difference in incidence between males and females in carcinomata of the arms and legs. The average delay to treatment is a year longer in carcinoma of the leg than in the other groups. It is noteworthy that the median age incidence is ten years younger in carcinoma of the lower extremity than in carcinomata of the upper extremity.

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A high percentage of the patients (45 per cent) gave a history of preexisting lesions (Table II). Senile keratoses preceded the development of cancer in 63 instances. Arsenical keratoses were present in 14 cases. Injuries or scars preceded the development of cancer in 36 cases. In addition to cancerous degeneration of traumatic scars, there were 23 cases with cancer developing in the scar of the burn. Radiation dermatitis was present as a precancerous lesion in ten cases, and tar dermatitis was present in four cases. Chronic osteomyelitis sinuses were recorded in 11 cases, and varicose ulcers in 11 cases. In two cases, postphlebitic ulcers underwent cancerous degeneration. A few patients definitely traced their cancers to preexisting skin conditions such as eczema, psoriasis, nevus, lupus, or gumma. Callus or clavus preceded cancer in three cases, and decubitus ulcer in two cases.

TABLE II

NATURE OF PRECANCEROUS LESIONS

Precancerous Lesion	Hand and Finger	Arm	Foot	Leg	Total
Keratosis	59	2	2	0	63
Arsenical keratosis	10	0	1	3	14
Injury and scar	23	2	5	6	36
Burn scar	5	7	2	9	23
Radiation dermatitis	8	0	0	2	10
Tar dermatitis	3	1	0	0	4
Osteomyelitis sinus	0	1	1	9	11
Varicose or postphlebitic ulcer	0	0	1	12	13
All others	8	2	5	4	19
Totals	116	15	17	45	193

Treatment.—The treatment of the local lesion was chiefly surgical. Radiation was successfully employed in many of the smaller lesions, often without pathologic confirmation of the diagnosis. Undoubtedly, many of the failures were due to inadequate treatment, and most of these patients were subsequently cured by surgery.

Carcinoma of an extremity should be locally curable in all cases, provided the treatment employed is sufficiently radical. Failure of local cure is primarily due to failure to appreciate the gravity of the process and the extent of the disease. Failure of local cure may also be attributable to the presence of remote, incurable matastases which makes the employment of radical measures futile, to refusal of the patient to submit to radical surgery, or to precarious general condition of the patient which may contraindicate appropriate local treatment. Table III presents the cases of known failure of cure of the local lesion.

TABLE III

FAILURE OF LOCAL CURE

Location	No. of Cases	Known Failure of Local Cure
Finger	30	3
Hand	205	18
Arm	27	4
Foot	24	3
Leg	46	4
Totals	332	32

Carcinoma of the Finger.—There were 33 cases of carcinoma of the finger, 3 (9 per cent) of whom presented or developed axillary node metastases. All 3 presented large lesions, in 2 cases of long duration and of low grade. One case was of short duration and grading was not carried out.

Carcinoma of the Hand.—Of the 205 hand carcinomata in which information is available, 30 (15 per cent) had or developed metastases. Eighteen of these patients had the lesion graded, of which 9 were of low grade, and 9 were of higher grade. Twenty-seven of 28 lesions were large, only 1 was small; 22 were of long duration, and 6 were of short duration. It is evident that the likelihood of node involvement increases with higher grades of malignancy, and with larger lesions and those of long duration. Only 2 patients without palpable lymph nodes on admission subsequently developed regional metastases. Both of these lesions were of high grade; 1 was small, and 1 was large. In one of these cases there was failure to control the local process as well. Neither patient was submitted to regional dissection, and both died.

Carcinoma of the Arm.—There were 27 cases of carcinoma of the arm in which information is available, of which 7 (26 per cent) presented regional metastases. All were large lesions of long duration, and the 4 which were graded were all of high grade malignancy.

Carcinoma of the Foot.—There were 24 cases of carcinoma of the foot in which information is available. Ten of the patients (41 per cent) with carcinoma of the foot presented regional lymph node involvement; and in all these cases the nodes were clinically enlarged on admission. Of the lesions with positive nodes, 7 were graded, and all proved to be of high grade of malignancy. Five were of short duration, and 5 were of long duration; 7 were large, and 3 were small. It is evident the size and duration here do not play such a conspicuous part in determining the likelihood of metastasis; while higher grades of malignancy seem to be of considerable importance.

Carcinoma of the Leg.—There were 46 cases of carcinoma of the leg in which information is available. Sixteen patients presented involvement of the regional nodes (36 per cent), and in all these cases the nodes were involved on admission to the hospital. Of the lesions associated with involved nodes, 12 cases were graded, of which 4 were low grade, and 8 were high grade; 12 were of long duration, and 4 were of short duration. Fourteen lesions were large, and 2 were small. Grade, size, and duration are all important in determining the probability of metastasis.

In summary of these groups of cases, it is evident that grade of malignancy, size, and duration of the lesion all have significant bearing on the likelihood of development of regional node involvement.

Table IV presents the incidence of regional node metastases from primary carcinoma in the various areas.

Clinical Appraisal of Lymph Node Involvement.—In the following analysis, nodes were considered to be free from metastasis when they showed no sign of involvement histologically, or when cure was effected by treatment of

the local lesion alone. Likewise, nodes were considered to be involved when they showed histologic evidence of metastasis, or when in advanced stages of the disease the clinical diagnosis of metastasis was obvious.

TABLE IV

INCIDENCE OF LYMPH NODE INVOLVEMENT

Location	No. of Cases	Node Metastasis	Percentage of Node Metastasis
Finger	33	3	9
Hand	205	30	15
Arm	27	7	26
Foot	24	10	41
Leg	46	16	36
Totals	335	66	

Among 154 patients with carcinoma of the upper extremity associated with nodes less than 1 cm. in diameter, actual metastasis was present or developed in 16 cases (11 per cent), and was absent in 138 cases. When nodes 1 to 2 cm. in size were present at the time of dissection, 5 cases (20 per cent) presented actual metastatic involvement, while 20 cases proved to be free from axillary disease. When nodes over 2 cm. in size were present they proved to be involved in 25 cases (86 per cent) and uninvolved in only 4 cases.

In the cases of carcinoma of the lower extremity, of 35 cases without appreciably enlarged nodes, metastasis was present in 5 (14 per cent). Fifteen patients presented nodes 1 to 2 cm. in size, and among these, metastases were present in 9 cases (60 per cent). Eighteen patients presented nodes over 2 cm. in diameter, and 17 of these harbored metastases. One patient who at the time of groin dissection proved to be free from metastasis, subsequently developed fatal recurrence in the groin area. The incidence of metastatic involvement in relation to the size of lymph nodes is shown in Table V.

TABLE V

INCIDENCE OF METASTATIC INVOLVEMENT IN RELATION TO SIZE OF LYMPH NODES

Location	Size of Nodes	No. of Cases	No. of Cases with Metastases	Percentage with Metastases
Axillary	0–1 cm.	154	16	11
Axillary	1–2 cm.	25	5	20
Axillary	Over 2 cm.	29	25	86
Inguinal	0–1 cm.	35	5	14
Inguinal	1–2 cm.	15	9	60
Inguinal	Over 2 cm.	18	17	94
Epitrochlear	0–1 cm.	15	3	20
Epitrochlear	1–2 cm.	11	8	72
Epitrochlear	Over 2 cm.	7	7	100
Totals		309	95	

There is a definite possibility of metastatic involvement even when nodes are impalpable or small, and when nodes are larger than 1 cm. the probability of metastasis is greatly increased. The relatively low incidence of involvement in axillary nodes, 1 to 2 cm. in size (20 per cent) is probably due to the inflammatory enlargement of nodes in many instances of hand carcinoma with infected ulcerations. It is evident that clinical appraisal of lymph node size

and involvement is more accurate in examination of the groin than it is in examination of the axilla.

Only one patient in the entire series developed metastasis to the popliteal lymph node area, and in this patient the involvement was clinically obvious, and recurrent disease proved to be fatal notwithstanding dissection.

Epitrochlear lymph nodes were described in 33 cases. The nodes were less than 1 cm. in size in 15 cases, of which only 3 (20 per cent) showed metastases. In 11 cases the nodes were 1 to 2 cm. in size, and of these, 8 (72 per cent) harbored metastases. Seven cases presented nodes over 2 cm. in size, and all proved to be cancerous. The statistics indicate that dissection should be carried out in any case in which the nodes in this area are palpable.

Efficacy of Lymph Node Dissection.—There was no fixed policy in regard to management of the regional lymph nodes. In most cases, small lesions without palpable regional lymph nodes received no primary treatment directed to the nodes. These cases were kept under observation, and later dissections were carried out if nodes appeared. Patients with more extensive local lesions were often subjected to primary dissection of the regional nodes, even in the absence of palpable metastatic involvement, as a prophylactic procedure. The dissections were usually performed at the same time as treatment to the primary lesion, and in other cases deferred for a few weeks. The dissections varied from simple excision of involved nodes to a block dissection of the entire lymph node drainage area.

In attempting to appraise the efficacy of lymph node dissection, it is arbitrarily assumed that patients who were free from any evidence of disease for two years or longer after treatment represented a "cure." This is desirable because otherwise it would be necessary to exclude as inconclusive a considerable number of cases in which the patients were untraced after two years and the results in which are of value in the inquiry into the adequacy of operation. The statistics, obviously, have no standing as an end-result study, and should not be so considered.

Axillary dissection was performed in 44 cases and the nodes proved to be involved in 21 instances. Nine cases were "cured," 11 died of recurrence, and 1 case was untraced and inconclusive. Thus, "cures" were obtained in 45 per cent of the 20 traced patients in whom axillary metastases were removed by lymph node dissection. There was one operative fatality among the cases submitted to axillary dissection.

The epitrochlear area was dissected in 16 cases, and the nodes were involved in 11 of these. One case was untraced and inconclusive, 7 were cured, and 3 patients ultimately succumbed probably because of failure to control the coincident axillary involvement.

Supraclavicular dissection was carried out in one case in which the nodes were obviously involved, but the patient succumbed to recurrence.

Groin dissection was performed in 28 instances, and nodes proved to be involved in metastases in 14 of these. Two patients were untraced, and hence inconclusive; 2 were cured, and 10 developed recurrence notwithstanding

dissection. Eight of these 10 represented delayed cases, in which dissection was not part of the original plan of treatment, but in which subsequent development of obvious clinical metastasis necessitated the operation. One patient who was submitted to a second groin dissection for recurrence was "cured." There was one operative fatality in the group submitted to groin dissection, who died as a result of acute cholecystitis which developed during the convalescence.

The popliteal area was dissected in two cases. In one of these the nodes were involved, and the patient succumbed to recurrence. The case in which the nodes were not involved could not be traced.

Roentgenotherapy was employed in a considerable number of patients with node involvement, in which the disease was inoperable or in which the patient's condition did not warrant dissection. In no instance was a cure obtained by this means.

Time of Appearance of Node Involvement.—Knowledge is available in regard to appearance time of axillary nodes secondary to hand and arm malignancies in 45 cases, and of inguinal nodes secondary to foot and leg cases in 23 cases.

TABLE VI

TIME OF APPEARANCE OF NODE INVOLVEMENT IN RELATION TO DURATION OF PRIMARY DISEASE

Metastasis Present After Onset of Primary Lesion	Per Cent of All Known Axillary Nodes	Per Cent of All Known Inguinal Nodes
Less than 6 mos.	22	40
Less than 1 yr.	36	52
Less than 18 mos.	48	56
Less than 2 yrs.	73	60
Less than 3 yrs.	75	65
Over 3 yrs.	25	35

In general, 75 per cent of axillary metastatic involvements, and 65 per cent of inguinal metastatic involvements, are present within three years after the onset of the primary tumor. Bearing in mind that the mean delay from onset to treatment of the primary lesion is 1.7 years in the upper extremity cases, and 2.8 years in the lower extremity cases, it is evident that a 2 to 3 year follow-up period after treatment should permit the detection of practically all regional metastases.

Metastases in Relation to Etiology.—None of the 11 patients with carcinoma originating in consequence of chronic osteomyelitis developed regional node involvement. There were 7 instances (30 per cent) of metastasis from carcinomata originating in burn scars, 4 (30 per cent) from carcinomata in varicose ulcers, 3 (21 per cent) from arsenical cancer, and 1 each in carcinomata originating in lesions of syphilis and lupus. Thus, with the exception of osteomyelitis, these precancerous lesions seem to give rise to cancers with a considerable likelihood of metastasis formation.

Duration of Disease in Fatal Cases.—Further knowledge in regard to needful follow-up period may be derived from the length of life after treatment, in cases who succumbed to recurrence. There were 41 cases who died

of recurrence of carcinoma of the upper extremity, in whom the time of death could be accurately determined. Twenty-three of these were dead within one year of treatment, and 37 within two years. Only 10 per cent survived longer than two years. Similar data are available in 27 cases of carcinoma of the lower extremity. Twenty-three of these patients had died within two years, and 26 within three years of the date of treatment. These statistics again emphasize that a relatively short follow-up period after treatment is probably sufficient for most cases, and that late manifestation of recurrence is exceptional.

Discussion.—Carcinomata of the extremities are, in the main, slowly growing, and of a low grade of malignancy. Failure to cure the local lesion is frequent and should be avoidable. The likelihood of metastasis is increased with longer duration, larger size, and higher grades of malignancy.

The size of regional nodes is not dependable as a guide to the presence or absence of metastasis. Small and inconspicuous nodes may harbor metastases in a definite number of cases. These metastases occur chiefly in association with large lesions of high grade of malignancy and of long duration. With larger nodes the likelihood of metastasis is greatly increased. Metastasis is very common in lesions involving the arms, legs, and feet. Metastasis is also common when a recognizable precancerous condition has been present for a considerable time. The conspicuous exception to this statement is carcinoma secondary to osteomyelitis, in which no metastases were observed.

Axillary and epitrochlear dissections are followed by cure in a considerable number of instances in which metastases occur. On the other hand, cures following dissection of inguinal metastases are relatively infrequent. It is probable that earlier and more thorough dissections would improve the results to be secured in these cases. There is insufficient data to permit a comparison of the efficacy of primary with delayed dissection. It is probable that if regional lymph node dissection is carried out as soon as node enlargement becomes evident the results will be nearly as good as when prophylactic dissections are employed. The policy of watchful waiting involves frequent and conscientious follow-up observation, and depends upon the complete and intelligent cooperation of the patient.

Lesions which are likely to develop metastases usually present them within a few years of the onset of the primary lesion. Late appearance of regional metastasis, especially after the eradication of the primary disease, is infrequent. Untreated and recurrent cases go on in a matter of a few years to a fatal termination, and late recurrence is relatively rare. For these reasons, it is perhaps not so necessary to subject these patients to the protracted follow-up observation period which is so important in some other types of malignant disease.

CONCLUSIONS

On the basis of the findings, treatment of the primary lesion must be prompt and thorough. Treatment of the lymph node area should be deferred

until after extirpation of the primary disease. When enlarged nodes persist after removal of the primary growth, regional dissection must be carried out as part of the initial treatment.

Differences of opinion will exist as to whether prophylactic dissection is justifiable in the absence of palpable lymph nodes. The high incidence of metastasis in carcinomata of the arm, leg, and foot (except for those cases associated with osteomyelitis) justifies the performance of prophylactic dissection in these cases, when the lesion is of any considerable extent. Likewise the high incidence of metastases in cases originating from a preexisting precancerous lesion (again except for osteomyelitis) strongly argues for prophylactic lymph node dissection. Primary dissection may be omitted in the absence of palpable nodes, in carcinomata of the fingers and hand, provided that the patient can be kept under close and strict surveillance for at least three years. During the first six months after operation, the patient should be observed at least once a month. During the next two years probably once in two months is often enough. Exceptionally, in poor risk patients, primary dissection may be omitted in some of the other groups of patients without palpable nodes, when there is opportunity for close surveillance.

Careful observation should be directed to the epitrochlear region as well as to the axilla, in cases with carcinoma of the upper extremity. Dissection of this area should be carried out if the nodes are palpable, and axillary dissection is indicated in all patients with epitrochlear node metastases.

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HYPOPROTHROMBINEMIA: EFFECT OF PERORAL AND PARENTERAL ADMINISTRATION OF A SYNTHETIC VITAMIN K SUBSTITUTE (2-METHYL-1, 4-NAPHTHOQUINONE)*

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When the value of alfalfa concentrates rich in vitamin K, used in the hemorrhagic disease of obstructive jaundice, was discovered, attempts were made to purify the extracts and isolate the active principle. As purer concentrates were prepared, insight into the chemical nature of the vitamin was rapidly obtained. In 1938, Almquist[1] pointed out that the then generally known properties of the vitamin indicated it to be a complex unsaturated hydrocarbon of low melting point. A little later, McKee and his associates[2] suggested that the vitamin had a quinoid structure. The first synthetic compound, shown to have vitamin K activity by Almquist and Klose,[3] was phthiocol (2-methyl, 3-hydroxy-1, 4-naphthoquinone), isolated from the tubercle bacillus, in 1933, by Anderson and Newman.[4] The first synthesis of vitamin K_1 was by Almquist and Klose,[5] who tentatively identified it as 2-methyl-3-phytyl-1, 4-naphthoquinone. Just previous to this, Ansbacher and Fernholz[6] reported upon the remarkably high vitamin K activity of 2-methyl-1, 4-naphthoquinone. This quinone has since been found to be so much more active (about four times as much) than vitamin K[7, 8] that it has led workers[9] to suggest that one unit of vitamin K activity be defined as the antihemorrhagic activity of one microgram of 2-methyl-1, 4-naphthoquinone. In spite of its high potency, the low solubility of this quinone, added to its irritating qualities when given parenterally, have restricted its usefulness somewhat. Attempts to find a product less irritating and more soluble yet retaining the high vitamin K activity of that quinone have, so far, proved fruitless. Toxicologic studies in animals have shown that the quinone possesses some toxic properties, though to obtain such effects amounts considerably higher than the therapeutic dose are required.[10] The crystalline quinone forms a yellow powder, well soluble in oil but soluble only one part in 10,000 of water at room temperature, and in a proportion of two and one-half parts to 10,000, if the solution is warmed to body temperature

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Methods.—The quinone* was given to patients perorally in the form of corn oil capsules (each containing 1 mg. of the drug), or intravenously in normal saline solution; in the latter case 10 mg. of the drug was dissolved in 50 cc. of hot physiologic salt solution just before the injection. For convenience, 10 mg. quantities were weighed out, placed in dark amber glass ampules, sterilized (120° C. for 30 mins. at 15 lbs. pressure), sealed and kept in a dark place. When it was desired to use the material the contents of one ampule was dissolved in 1 cc. of absolute ethyl alcohol and slowly poured into 50 cc. of warm sterile physiologic salt solution in a flask wrapped in black paper. The instability of the drug in watery solution makes these precautions desirable.

Plasma prothrombin was measured by the method of Quick.[11] Each reading represents the average of four simultaneous determinations on the same specimen. The average of the pooled times of three normal plasmas was used as representing 100 per cent prothrombin. Whenever possible the same three normal young adults were used for this purpose, blood (2.25 cc.) being collected from them regularly three times a week for as long a period as two months, continuously. These long periods of observation disclosed the fact that some individuals have a prothrombin concentration at a constantly higher (or lower) level than other normal individuals, a point to be discussed in greater detail elsewhere.

TABLE I

INTRAVENOUS ADMINISTRATION OF 2-METHYL-1, 4-NAPHTHOQUINONE

No.	Patient	Diagnosis	Prothrombin % Before	Prothrombin % After	Amt. Given Mg.	Time Interval (Days=d. Hrs.=h.)	Bile Salts Gm.	Bleeding Site	Remarks
1	F. McG.	Cholecystectomy; bil. fistula	20	55	2	3 h.	0	Wound	Bleeding ceased
2	M. T.	Carcinomatosis, abdominal	10	80	6	5 d.	0	Urin. bladder	3 injections; bleeding stopped after 1st injection
3	M. C.	Carcinoma of the rectum	27	49	2	3 d.	0	Skin	Colostomy
4	E. G.	Cirrhosis of the liver	40	37	2	4 h.	0	0	Ser. bilirubin, 2.3 mg.
5	F. C.	Cirrhosis of the liver	45	42	2.5	2 h.	8	0	No subseq. rise. Ser. bilirubin = 3.0
6	Bar.	Carcinoma of tongue	46	44	2	2 h.	0	Ulcer of tongue	Left hospital 2 days after
7	Tib.	Lung abscess	43	45	2	2 h.	0	0	No subseq. rise
8	S. M.	Fatty liver; alcohol. cirrh.	44	48	9	9 d.	0	Gums	Ser. bilirubin = 1.2; 6 inj.
9	F. M.	Lung abscess	55	100	2	7 h.	0	0	No rise on oral administration previously

Results.—In Table I are listed the changes in the plasma prothrombin of various patients who received the quinone intravenously. In only three

* The 2-methyl-1, 4-naphthoquinone in liquid oil, in oil capsules or in powder was supplied by Dr. A. Black of the Squibb Research Institute.

of these patients was the prothrombin below 30 per cent of normal; in two of them (Nos. 1 and 2) there was a prompt and favorable response accompanied by diminution and disappearance of the signs of bleeding, and in the third (No. 3), the bleeding diminished, but it was not possible to measure the prothrombin until three days after the injection, when a significant rise was observed. A change from 20 to 55 per cent was detected as early as three hours after an injection in a patient (No. 1) bleeding from a wound, following a cholecystectomy. In another patient (No. 9), with a lung abscess occupying almost the entire right lung, the plasma prothrombin increased from 55 to 100 per cent within seven hours after an injection.

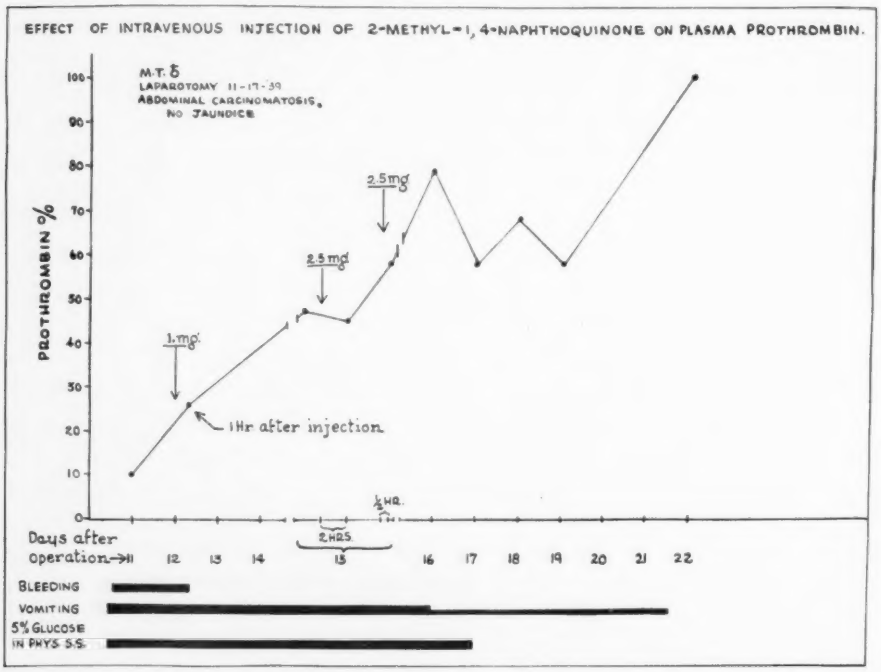

CHART 1.—Arrows indicate time of injections.

Chart 1 illustrates the increase in the plasma prothrombin after intravenous injection of 2-methyl-1, 4-naphthoquinone in a patient who had been vomiting almost continuously for 11 days following a celiotomy. Just before the administration of the synthetic compound, she had been passing large amounts of blood in the urine, as obtained by catheter. Coincident with the rise of the plasma prothrombin the bleeding stopped and did not recur. In all others listed in Table I the drug was administered intravenously, not on account of the level of the plasma prothrombin or because of manifestations of bleeding, but as a test of the power of the drug to raise the level of the prothrombin from moderately low concentrations, and as a preparation for a surgical procedure or a paracentesis. The plasma prothrombin in these patients was between 40 and 50 per cent, and in none of

them was there a significant, immediate or delayed rise after the injections, amounting in one patient (No. 8) to as many as six for a total of 9 mg. of the quinone

Chart 2 illustrates the changes in the plasma prothrombin concentration in a woman with advanced, alcoholic fatty cirrhosis of the liver. This patient during a prolonged hospital stay, not indicated on the table or in the chart, received by mouth large amounts of the various potent natural vitamin K concentrates as well as the quinone, with or without bile salts.

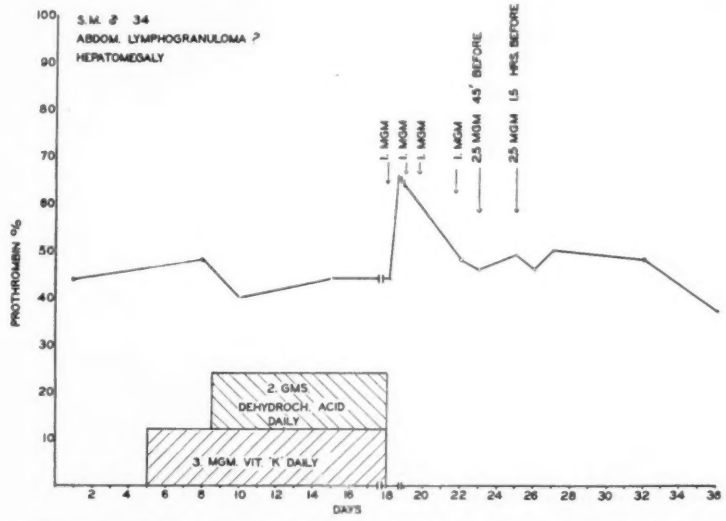

CHART 2.—Effect of peroral (blocked area) and intravenous (arrows) administration of 2-methyl-1, 4-naphthoquinone. At autopsy, it was demonstrated that the patient had alcoholic, fatty metamorphosis of the liver. The provisional clinical diagnosis of abdominal lymphogranuloma had been made during life.

"Klotogen" and "Cerophyl," and as much as 9 mg. of the quinone per day was given to the other patients who were refractory to the therapy in the usual doses. In patients with suppurative diseases of the lung, a hypoprothrombinemia is an almost constant finding, and it appears that it is only with large doses of the vitamin or its substitutes that the deficiency may be overcome.

In the group of patients with hepatocellular disease (Nos. 4, 5 and 8), intravenous administration could not, of course, be expected to correct the fundamental cause for the hypoprothrombinemia, namely interference with production of prothrombin by the liver. Vitamin K therapy, combined with plasma or whole blood transfusions, might have been more effective in raising the prothrombin level in this group, the introduced blood supplying materials other than vitamin K required for the production of prothrombin. With one exception, none of these patients exhibited any signs of bleeding even during or after extensive operations (*e.g.*, resection of the sigmoid). In this group the blood prothrombin level does not seem to constitute as sensitive an indicator of a tendency to bleed as in the hypo-

prothrombinemias due to a vitamin K deficiency. The possibility remains that the 40–50 per cent levels found in this group do not truly express the amount of plasma prothrombin in these patients; the method employed for measuring prothrombin may be influenced by qualitative changes in this substance and thus yield, sometimes, a confusing result.

There were no manifestations at any time that could be interpreted as toxic effects of the drug. A few patients complained of a burning sensation along the course of the vein while the injection was being given.

TABLE II

ORAL ADMINISTRATION OF 2-METHYL-1, 4-NAPHTHOQUINONE

No.	Patient	Diagnosis	Prothrombin % Before	Prothrombin % After	Amt. Given Mg.	Time Interval Days = d.	Bile Salts Gm.	Bleeding Site	Remarks
1	J. M.	Incomplete abortion	22	78	6	3 d.	0	Menorrhagia	Bleeding abated before D. and C.
2	R. M.	Obstructive jaundice	25	200	4	2 d.	0	Skin hematoma	Bleeding ceased
3	Tib.	Lung abscess	40	43	8	4 d.	0	0	No subseq. rise
4	S. M.	Fatty liver; alcohol. cirrh.	48	44	30	10 d.	14	Gums	
5	R. C.	Abdominal lymphogranuloma	45	47	7	7 d.	10	Skin	Provis. diagnosis
6	E. P.	Portal cirrhosis	22	36	7	7 d.	4	Skin	Ser. bilirubin = 23
7	E. P.	Portal cirrhosis	36	50	21	7 d.	0	Skin	Ser. bilirubin = 26
8	C. B.	Carc. rectum; liver metast.	25	43	54	17 d.	0	0	No unusual bleeding at oper.
9	C. H.	Carc. liver; metastatic	24	100	4	4 d.	0	0	Ser. bilirubin = 22
10	G. C.	Coronary art. dis.	57	90	4	4 d.	0	Bowel	Bleeding stopped
11	A. R.	Prostatectomy; hyper. prostate	50	93	27	9 d.	0	Wound	Bleeding stopped
12	E. M.	Gastric resection	10	90	2	2 d.	0	Bowel	Bleeding stopped
13	L. W.	Cholecystotomy; cholelithiasis	22	100	2	2 d.	0	Wound	Bleeding stopped
14	F. C.	Portal cirrhosis	37	48	21	7 d.	14	0	
15	N. C.	Cholecystectomy; cholelithiasis	50	70	10	10 d.	0	0	No jaundice
16	H. H.	Gastrocolic fistula	40	120	25	5 d.	0	0	
17	F. N.	Carc. rectum; metast. liver	60	55	15	3 d.	0	0	
18	E. G.	Cirrhosis of liver	42	43	28	4 d.	0	0	Ser. bilirubin, 2.3 mg.
19	F. M.	Lung abscess	45	55	16	2 d.	0	0	
20	Baby B.	Hemorrh. disease; newborn	<1	100	0.3	1 d.	0	Internal ear; puncture wounds	Bleeding stopped 2 hrs. after giving 0.1 mg.

In Table II are listed the changes produced by the oral administration of the quinone in liquid corn oil or in corn oil capsules. The impression was obtained from the diminution in the bleeding, that in those patients who could retain the material by mouth, almost as prompt a rise in the plasma prothrombin took place as after intravenous administration. The

group that proved refractory to therapy was again that in which the prothrombin was generally between 30 and 50 per cent of normal to begin with, and in whom hemorrhagic manifestations were seldom found.

Patient E. M. (Chart 3), who had undergone a subtotal gastrectomy for gastric and duodenal ulcers, and was vomiting most of his liquid feedings, had to be given several blood transfusions to replace the blood lost in the bowel. The prothrombin eight days after the operation was 10 per cent. Administration of 1 mg. of the quinone by mouth raised the prothrombin

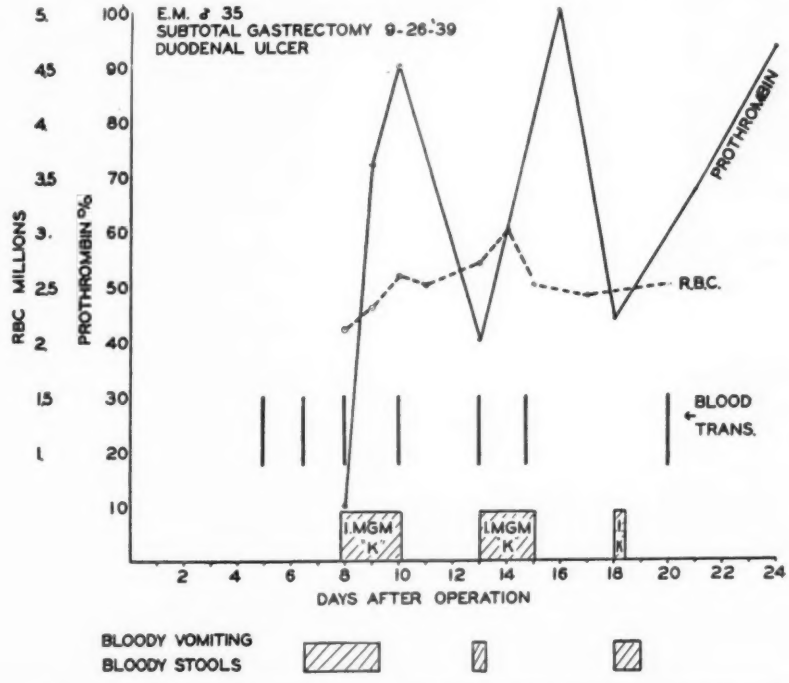

CHART 3.—Effect of blood transfusions (heavy vertical lines) and peroral administration (blocked areas) of 2-methyl-1, 4-naphthoquinone. The drug was purposely withdrawn at intervals to note the effect on bleeding and plasma prothrombin. Dose in milligrams per day indicated in the area.

to 72 per cent of normal in 24 hours. It was felt that in this, and other patients reacting in an analogous manner, ingestion of the drug followed by a blood transfusion led to more rapid and favorable results than by the drug alone. Blood transfusions alone are known to produce only a small and transient rise in prothrombin.[12]

Patient L. W. (Chart 4), who underwent a cholecystotomy, is another example of the same type of reaction. Both of these patients must have been entirely depleted of the vitamin by their illness and postoperative vomiting, for, though a prompt rise followed ingestion of as little as 1 mg. of the quinone, the high prothrombin level was not maintained when the drug was withdrawn. Table II serves also to illustrate the variety and number

of clinical conditions in which a hypoprothrombinemia is responsible for what may constitute the most important complication of the disease—excessive bleeding. Patient J. M. (No. 1), for example, developed a hypoprothrombinemia following an infected, incomplete abortion; the bleeding stopped promptly after therapy with the synthetic quinone, to recur when its administration was discontinued temporarily. How a vitamin K

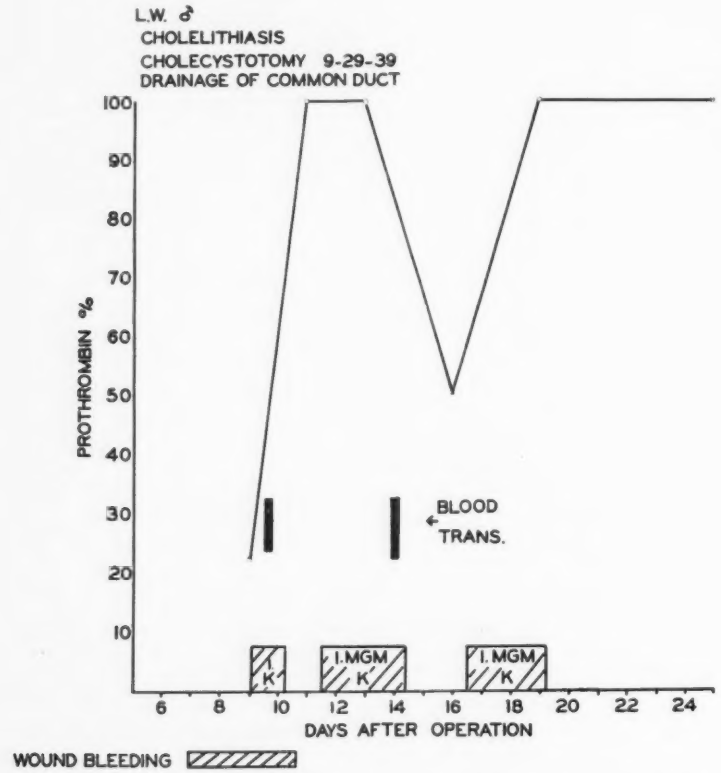

CHART 4.—Effect of blood transfusions and peroral administration of 2-methyl-1, 4-naphthoquinone on plasma prothrombin.

deficiency developed in this and other similar cases is not, at present, apparent.

SUMMARY

(1) In 23 patients with various grades of hypoprothrombinemia, 29 trials of the effect of oral or intravenous administration of 2-methyl-1, 4-naphthoquinone were carried out

(2) A dose as small as 1 mg. per day, in an adult, may cause a prompt, substantial rise in the plasma prothrombin from low levels, and check all hemorrhagic manifestations. Higher doses appear necessary to produce an effect when these small doses fail.

(3) After intravenous administration, a significant rise may be detected as early as three hours after an injection. No signs of a toxic effect were observed.

(4) A group of moderately hypoprothrombinemic patients, chiefly with hepatocellular disease, appear refractory to therapy with the quinone as well as with natural vitamin K.

(5) Blood transfusion combined with therapy with the quinone may produce quicker results than the quinone alone.

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THE LOCAL USE OF SULFANILAMIDE, SULFAPYRIDINE AND SULFAMETHYLTHIAZOL*

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Our interest in the local implantation of sulfanilamide powder was aroused by the results obtained in the treatment of compound fractures at Doctor Zierold's clinic, in Minneapolis. This work, published by Jensen, Johnsrud and Nelson,[1] showed that the local implantation of sulfanilamide powder in the wound before suturing the débrided compound fracture reduced the incidence of infection at that hospital from 27 to 5 per cent. We not only adopted this method of treating compound fractures, but, since we had been disappointed in the effect on local lesions after the oral administration of sulfanilamide, we have been using the drug locally in infections of soft tissues and of bone, and have used it prophylactically in certain clean operations where there was considerable tissue damage or where foreign material was left in the wound. We have also used it routinely in wounds after operations on tissues which had previously been infected and in operations near infected tissue, such as amputations for infection and plastic operations for chronic osteomyelitis. The clinical results have been satisfactory.

We, naturally, wanted to know whether or not the drug damaged the tissues or interfered with wound healing. We also have been troubled concerning the sterility of the drug, because, when we put foreign material in a clean operative wound, we want to be sure that the material is sterile, or at least that it will be bactericidal. It is well-known that the results obtained in animals with sulfanilamide cannot be duplicated in the test tube. This is due to two factors: (1) Sulfanilamide in the dilute concentrations which are obtained in the blood is merely bacteriostatic for susceptible organisms and the bacteria must then be destroyed by the clearing mechanism of the infected animal. In the test tube there is no clearing mechanism and eventually the bacteria begin to grow; and (2) the action of sulfanilamide is inhibited by small amounts of peptone and peptone-like substances in culture media and most culture media contain peptone (Lockwood[2]). If these substances are omitted from the media and the number of bacteria is small, then the results of an *in vitro* experiment approach those obtained *in vivo*, but thick suspensions of bacteria give off substances which inhibit the action of sulfanilamide and of sulfapyridine (Fleming[3]).

It is further to be noted that the efficiency of sulfanilamide varies directly with the concentration of the drug which comes in contact with the bacteria and inversely with the number of bacteria present. When we put sulfanilamide powder in a wound, the serum which collects in the wound will become sat-

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urated with the drug. As this is soluble up to about 1 per cent at body temperature, the concentration obtained in the wound is almost 100 times that which is obtained in the blood after therapeutic doses. This concentration of the drug is constant until the drug is absorbed and is not limited to the wound, but extends into the surrounding tissues, as can readily be demonstrated by placing "prontosil soluble" in a wound and examining the wound 12 hours later. The red dye diffuses into and stains the tissues for a considerable distance around the wound.

When the concentration of sulfanilamide is increased to near the saturation point, its range of activity is greatly broadened and we are no longer dealing with a drug which is merely bacteriostatic for certain *Beta*-hemolytic streptococci and a few other susceptible bacteria, but we have a drug which affects a wide variety of organisms. Among these are staphylococci and C. welchii (Spink,[4] and Sadusk and Manahan[5]). Also, the drug is no longer merely bacteriostatic, but is actually bactericidal if the organisms are not too numerous and if the culture medium is favorable to the action of the drug.

In our wounds, we have a test tube experiment with a saturated solution of the drug. This is modified by certain factors. The first of these is that we have a culture medium which is unfavorable to the sulfanilamide, because tissues break down into peptone-like substances. King and Henschel[6] have shown that the products of tissue disintegration inhibit the action of the drug. The other two factors are favorable, because sulfanilamide neutralizes the toxins of staphylococci and C. welchii (Carpenter and Barbour[7]) and thus limits the amount of tissue breakdown.

In addition to the above, we have in the wound the normal clearing mechanism of the animal and apparently phagocytosis occurs and tends to get rid of the few organisms which are left and which are prevented from growing. It is not known just what effect a saturated solution of sulfanilamide has on leukocytes, but apparently leukocytes and connective tissues are very resistant to the drug, and King[8] has shown that a concentration of 100 mg. per cent accelerates the migration rate of leukocytes.

In a previous publication (Key and Burford[9]) it has been shown that sulfanilamide powder in experimental fractures in animals does not measurably inhibit the union of the bone or the healing of the wound. On the other hand, Bricker and Graham[10] have shown that in animals in which a therapeutic concentration of sulfanilamide was maintained in the blood, experimental wounds in the stomach did not heal quite as firmly in a given length of time as did those in normal control animals.

Clinically, we have observed that some of the wounds in which we have placed sulfanilamide crystals have not healed normally; that is, it is not unusual for a considerable amount of dark red, bloody fluid to collect in these wounds. Usually this is absorbed without separation of the skin edges and the wounds heal *per primam*. Occasionally, it drains out through a small opening in the skin and then the wound heals, although somewhat more slowly than usual. We believe that this is because sulfanilamide interferes with the

formation of the clot. When the dry powder is mixed with normal blood it delays the clotting time and interferes with the formation of the clot. It is possible that this slight interference with the healing of the wound can be lessened by not putting as much powder in the wound as we have done in the past.

Since sulfanilamide is not entirely satisfactory, and since its general effect is limited to certain susceptible bacteria which are relatively few in number, chemists have produced new compounds related to this drug and have tried out the effect of these compounds on various types of organisms, both *in vitro* and *in vivo*. Among these, sulfapyridine has proved its value in the treatment of infections due to pneumococci, and there is some evidence that it is more active against staphylococci than is sulfanilamide.

Recently, a new compound, sulfamethylthiazol, has been produced and its use is now in the experimental stage. Barlow and Homburger[11] have shown that sulfamethylthiazol prolongs the life of mice infected experimentally with a virulent strain of *Staphylococcus aureus* and prevents the development and permits the healing of abscesses in kidneys and other organs in a significant number of animals, and Rake and McKee[12] have shown that sulfamethylthiazol *in vitro* is more active against *Staphylococcus aureus* than is sulfapyridine and that it protected mice from infection with staphylococci in a high percentage of the animals infected. McKee, Rake, Greep and van Dyke[13] have shown that when these compounds are administered in 1 per cent of the diet the effect of sulfathiazol is equal to that of sulfapyridine in protecting mice from infection with pneumococci. Lawrence[14] has shown that thiazol compounds in concentrations as low as 5 mg. per cent are superior to sulfanilamide and sulfapyridine in their inhibitory actions on pneumococci and *Beta*-hemolytic streptococci. The methyl and phenyl derivatives were found to be bacteriostatic for *Staphylococcus aureus* while sulfanilamide and sulfapyridine exhibited only a moderate degree of inhibition upon this organism. He used concentrations as high as 200 mg. per cent of each drug. Barlow and Homburger[15] concluded that the chemotherapeutic effects of three thiazol derivatives of sulfanilamide under conditions of experimental streptococcal or pneumococcal infections are definitely superior to those of the parent substance and compare favorably with those of sulfapyridine. They further state that on the basis of superior margin of safety of sulfathiazol, and particularly sulfamethylthiazol, as compared with sulfanilamide and sulfapyridine, these new compounds appear quite likely to be useful.

Long and Bliss[16] conclude that sulfathiazol is as effective a bacteriostatic agent as is sulfapyridine in broth cultures on certain strains of hemolytic streptococci and staphylococci, but is, in their experiments, slightly less effective than sulfapyridine in the control of experimental pneumococcal infection. Long, Haviland and Edwards[17] conclude that sulfathiazol and sulfapyridine have about the same acute toxicity in mice and that this is about one-third greater than that of sulfanilamide. The acute toxicity was measured by the subcutaneous injection of the soluble sodium salts of the thiazol derivatives.

Van Dyke, Greep, Rake and McKee[18] found that the toxicity of sulfathiazol was about 65 per cent that of sulfapyridine, but that repeated administrations of the drug in food of mice indicate that sulfathiazol is more toxic than sulfapyridine at a high dose-level, but that there is no difference at a dose-level which is effective therapeutically. With both drugs the principal pathologic change appeared to be renal damage.

Since most of the interest in sulfanilamide and its allied compounds has been from the standpoint of those who administer the drug in therapeutic doses and obtain concentrations of about 10 mg. per cent in the blood stream, most of the work which has been done on the bacteriostatic action of these drugs has been undertaken with low concentrations and relatively little has been accomplished on the effects of high concentrations of the drugs. From the standpoint of wound infection, we are interested in the effect of the drugs on hemolytic streptococci, staphylococci and C. welchii, and we are interested in the effect of saturated solutions of the drug on these organisms. Our first interest is whether or not these drugs are bactericidal for these organisms *in vitro*, and since our wounds may be assumed to contain some peptone-like substances we want to know whether or not the drugs will kill bacteria in culture media which contain peptone.

In order to determine this, we carried out a few experiments with supersaturated solutions of the drugs, using 24-hour peptone broth cultures of *Staphylococcus aureus*, hemolytic streptococcus and C. welchii. The drug was first placed in the culture tube in excess, so that some drug was left in the bottom of the tube and this was left in the incubator for 24 hours. The tubes were inoculated and placed in the incubator. Some tubes were inoculated with one loop of a 24-hour broth culture and others with four loops and others with 1 cc. of the culture. The C. welchii cultures were incubated under anaerobic conditions. Since sulfanilamide is soluble up to about 1,000 mg. per cent and the other two drugs are soluble to slightly less than 200 mg. per cent, the concentration of sulfanilamide in the culture media was about five times that of sulfapyridine or sulfamethylthiazol. On the other hand, that is the condition which would exist in a wound which contained the drug in excess. The tubes were examined for turbidity and by smears and subcultures at the end of 24, 48 and 72 hours. It was found that the saturated solution of sulfanilamide completely sterilized the tubes containing the streptococci, but that the staphylococci and the gas bacilli grew, although they were inhibited during the first 48 hours. In the tubes containing sulfamethylthiazol and sulfapyridine, all of the organisms grew at the end of 24 hours, but were inhibited.

In the above crude experiments no bacterial counts were attempted, but it was evident that in broth containing 2 per cent of tryptose and supersaturated with the drug, the growth of all three organisms was definitely inhibited during the first 48 hours. This was true of each drug and even with heavy inoculations of bacteria. None of the drugs were bactericidal for staphylococci or C. welchii, and only sulfanilamide was bactericidal for strep-

tococci. Consequently, we believe that the drugs should be autoclaved before they are used in wounds. This may cause the powder to form lumps, but these can be crushed when used.

Jensen, Johnsrud and Nelson[1] produced compound fractures of the ribs in a series of 27 guinea-pigs and flooded the wounds with a culture of *Staphylococcus aureus*. The wounds were then sutured with silk. In seven control animals, 65 per cent of the wounds became infected and osteomyelitis developed in the ribs. In ten animals, 0.5 Gm. of sulfanilamide was implanted beneath the skin of the abdomen. Eighty per cent of the wounds in these animals became infected. In ten other animals in which sulfanilamide powder was placed in the wound before the wound was sutured, primary healing occurred in 80 per cent of the animals.

It was our intention in beginning this work to repeat the above experiments and to determine the relative efficiency of sulfanilamide, sulfapyridine and sulfamethylthiazol in preventing infection in contaminated compound fractures of ribs in a series of guinea-pigs. Unfortunately, we were not able to repeat the experiment, although we used four different strains of virulent hemolytic *Staphylococcus aureus*. In our guinea-pigs there was a high mortality from the fractures of the ribs, as we frequently penetrated the pleura and the animal then died. In our first experiments, the wound was flooded with a 24-hour broth culture of the staphylococcus and about 3 gr. of sulfanilamide, sulfapyridine or sulfamethylthiazol was implanted in the wound and it was sutured with silk. In 12 animals, only two wounds broke open (one with sulfanilamide and one with sulfamethylthiazol). These drained for a few days and then healed. The three control wounds which contained the bacteria, but no drug, healed by primary intention and remained healed.

We then tried the same experiment with rabbits, using a rabbit-virulent strain of staphylococcus obtained from Doctor Julianelle. The broth culture of this was swabbed in the wound after the rib had been fractured. The wound was then sutured. There were six controls and six wounds each with sulfanilamide, sulfapyridine and sulfamethylthiazol. About 5 gr. of the drug was placed in the wound before it was sutured. In this experiment the bacteria were grown on blood agar plates and were washed off with a small amount of salt solution and then swabbed into the wound. On the fourth day one wound containing sulfapyridine and another, containing sulfanilamide, were found to be broken open; the other wounds all healed. It is believed that the relatively large amount of the powdered drug in these two wounds acted as a foreign body and caused them to break open in the two instances in which this occurred, as the controls all healed.

In another experiment with 12 guinea-pigs, compound fractures of the ulnae were produced and the wounds swabbed with cultures of *Staphylococcus aureus* and sutured. In these experiments acacia plus glucose was added to the 24-hour broth culture before swabbing the wound with the material. Three of these animals were used as controls. In the other nine, the three above

mentioned drugs were placed in the wounds. One wound which contained sulfanilamide broke open on the fifth day; the others all healed.

In a fourth experiment, compound fractures of the ulnae were produced in rabbits and the wounds were saturated with virulent hemolytic *Staphylococcus aureus* plus acacia and glucose and then sutured, as were the guinea-pigs above. In five rabbits so treated, all wounds healed except one. This broke open at the end of five days and drained for several days and then healed. None of these rabbits had any drug placed in the wounds.

It is evident from the above experiments that we did not feel justified in drawing any conclusions as to the relative effects of the drug used, because in each of these four types of experiments our control animals failed to develop osteomyelitis, or even to develop sufficient local soft tissue infection to cause the wounds to break open.

We then decided to investigate the manner in which the tissues of experimental animals reacted to a local implantation of each of the above drugs. In order to study this, operations were performed upon a series of dogs, under general anesthesia, and approximately 7 gr. of sulfanilamide or of sulfamethylthiazol or of sulfapyridine was implanted in the wound. The wound was then sutured and the animal was sacrificed later and the wound was inspected locally; it was then excised and sectioned; the sections were stained with hematoxylin and eosin and studied microscopically. In this experiment, each drug was implanted into a joint and into a muscle, and the experiment was controlled by similar wounds in similar joints or muscles of the same animals. The shoulder, hip, elbow and ankle joints were used, and the operations were so spaced that when the animals were sacrificed we had 12 specimens at intervals of from one to 15 days after the implantation of each drug, and 12 controls of the same period.

On inspection of the wounds, we were not able to determine that any of the drugs caused any more inflammation or any more necrosis in the wounds and in the healing tissues than was present in the controls, with the exception that the wounds which contained the drugs and which were examined during the first few days after the operation tended to contain more fluid than the controls, and blood clots in these wounds were more gelatinous and less firm than those present in the controls. It is also to be noted that most of the wounds healed *per primam*, although in a few the skin edges separated. This separation of the skin edges did not occur with any more regularity in the wounds which contained the drug than it did in the controls, nor could any particular drug be incriminated as being prone to cause separation of the skin edges. At the time of our inspection of the gross wound it was not possible to identify any of the powdered drug, even after the short interval of 24 hours.

The microscopic sections failed to reveal any constant difference between the control wounds and the wounds which contained the drugs, except in the 24-hour wounds containing sulfapyridine and sulfamethylthiazol, where evidence of a few crystals was found in the microscopic sections. In other wounds the drugs had disappeared and the tissues seemed to be healing in

the normal way. In some of these, even in wounds which contained the drugs, there was a low grade infection present around the deeper sutures. These wounds contained deep sutures of silk which tended to render them more susceptible to infection, and in performing the operations little care was taken to prevent infection, as we wanted to determine whether or not we could, in this way, obtain some idea as to the relative efficacy of the drugs.

In the joints there was a low grade inflammation of the synovial tissues as a result of the operative incision. However, this did not seem to be sufficient to damage the joint seriously. The synovial lining cells were somewhat more numerous and larger and more youthful in appearance, and there was a moderate amount of infiltration of round cells and leukocytes in the specimens examined during the first ten days after the operation. At the end of the tenth or twelfth day, however, the synovial lining cells had returned to approximately their normal appearance and the infiltration of cells in the subsynovial tissues had almost entirely disappeared. It is to be noted that this low grade inflammation in the lining of the joints and the subsynovial tissues was no more marked in the joints in which either sulfanilamide or one of the other drugs had been placed than it was in the joints which had simply been opened and sutured. In other words, whenever a joint is opened and sutured, one may expect such reactions to occur in the synovial tissues. There was no evidence that the drug caused any damage to the surface of the articular cartilage, even though in these experiments the powdered drug was placed directly in the joints of the animal and then the wound sutured so that a considerable quantity of the drug was left in the joint.

It is thus evident from this series of experiments that either sulfanilamide or one of the newer preparations—that is, sulfapyridine or sulfamethylthiazol—can be placed in the tissues with impunity and it may be expected that they will not seriously harm healing tissues or prevent healing.

In a second series of experiments we attempted to investigate the effects of these drugs when injected into joint cavities and into other tissues and body cavities. These experiments were performed upon rabbits, and the drugs were injected into the peritoneal cavity, pleural cavity, knee joints and the subcutaneous tissues. In these experiments we used supersaturated solutions of the drug, that of sulfanilamide being approximately 1,000 mg. per cent while those of the sulfapyridine and sulfamethylthiazol were in the vicinity of 200 mg. per cent. We would, of course, have liked to use more highly concentrated solutions, but could not do so on account of the poor solubility of the drugs. It should be mentioned that both sulfapyridine and sulfamethylthiazol can be obtained as the sodium salt, which is very soluble, but these salts are very alkaline, having a p_H of about 11, and consequently are very irritating to the tissues; and we would not expect them to be tolerated either subcutaneously or in the body cavities. Consequently, they were not used.

In order to obtain a more highly concentrated solution we also used neoprontosil in 5 per cent and 2.5 per cent solutions. These experiments were

controlled by the injection of an equal amount of normal salt solution. They lasted only two days; that is, at the end of 24 and 48 hours after the injection the animals were sacrificed, and pieces of the lung, areas of the subcutaneous tissue which had been injected, pieces of the omentum and the synovial lining of the knee joint were excised and studied microscopically. In none of the animals were we able on macroscopic examination to see that the injection had done any harm, other than an occasional area which had been injured by the injecting needle where there was a small hemorrhage. Apparently, the tissues tolerated these substances equally well.

On microscopic examination of the knee joints, it was found that the synovial lining cells exhibited slight swelling and there was a slight amount of proliferation of the lining cells and also a slight amount of infiltration of round cells and leukocytes in certain loose areas of the subsynovial tissues. The pleural and peritoneal surfaces and the subcutaneous tissues presented even less departure from the normal than did the knee joints. These slight changes were also noted in the tissues and cavities which received normal salt solution and had largely disappeared on the second day. On the second day, the neoprontosil, which had been injected into the knee joints in 2.5 and 5 per cent solutions, was practically all gone and only a faint pinkish tinge remained. At the end of the first day, the knee was quite pink and the pink dye was seen spread out in the tissues all around the knee joint for considerable distances. It is further to be noted that neoprontosil injected into the joint appeared in the urine of the animal within a few minutes after the injection.

We also tested the absorption of the drugs in the subcutaneous tissues. In a series of eight rabbits, four small incisions were made in the skin of the back. The subcutaneous tissues were separated and about 5 gr. of the powdered drug was pushed well back under the skin and the wounds were sutured. The animals were sacrificed on the second, third, fifth, seventh, ninth, twelfth, twenty-first and twenty-eighth days after the operation. The neoprontosil, being quite soluble, was absorbed promptly and the powder had almost disappeared by the second day and was replaced by a gelatinous-like area of inflammatory tissue which disappeared in a few days. The sulfanilamide, being moderately soluble, was about half gone on the second day and practically all of it had disappeared on the fifth day, and the mild inflammatory reaction in the area was less than with the neoprontosil. The sulfamethylthiazol and the sulfapyridine, being relatively insoluble, remained as round, button-like masses in the subcutaneous tissues and there was very little reaction around them. Apparently these drugs were being encysted or surrounded by capsules, and their absorption was going on very slowly. At the end of five days, the sulfamethylthiazol and sulfapyridine remained about as in the two-day animal, while the more soluble neoprontosil and sulfanilamide had completely disappeared. In the animals examined at longer intervals, the two less soluble drugs were encapsulated, so that only a small encysted mass was present at the end of four weeks. A comparison of these experiments with those in which the drugs were implanted in wounds in muscles and in joints, indicates

that the circulation in the tissues surrounding the implantation of the drug has a good deal to do with the rate at which the drug will be absorbed. The rate also varies inversely with the solubility of the drug.

CONCLUSIONS

(1) Sulfanilamide, sulfapyridine and sulfamethylthiazol, in the form of powder, are well tolerated by the joints, muscles and connective tissues.

(2) In solution, they are well tolerated by the joints, pleura and peritoneum.

(3) They are bacteriostatic, but not bactericidal under the conditions present in a wound and should be sterilized before they are placed in a clean wound.

(4) They slightly inhibit the early healing of the wound, but do not unduly prolong the period of healing.

(5) Our experiments do not permit us to evaluate the efficacy of the drugs in preventing infection in contaminated wounds.

(6) *In vitro* observations, in the literature, suggest that local implantation, which assures a temporary high local concentration of the drug, is an effective method of preventing infection in clean and in contaminated wounds and of treating open infected wounds. Our clinical experience indicates that this is true.

(7) Due to its greater solubility we prefer sulfanilamide powder to either of the other drugs.

(8) We advocate the local implantation of sulfanilamide powder not only in contaminated wounds, but also in clinically clean operative wounds where infection is especially to be feared or would be especially undesirable.

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DISCUSSION.—DR. KELLOGG SPEED (Chicago, Ill.): I should like to corroborate some of these findings and suggestions of Doctor Key. I have used sulfanilamide in some dozen clinical cases, and we have found that there is an increased amount of serous discharge from the wounds without a definite delay in the healing. I think the question of drainage of the wound has a lot to do with the keeping of the concentration of the drug locally, and we have found it of greatest use in secondary amputations, and in the removal of septic stumps where one wishes to close the area tightly in an attempt to obtain early healing. We take a small handful of the drug, without measuring its quantity, and place it directly in the closed stump. These have done very well with, as I said, prolonged serous discharge. We have found no evidence, clinically, of untoward reaction in the tissues of such wounds.

We have also used, in definitely septic wounds which may contaminate bone or joint, irrigations of 4 per cent solution of sulfanilamide. This we find reduces the bacterial count, but does not completely sterilize the wound in any way. It does seem, in cases that have been observed as long as three months, to minimize the amount of new bone formation or osteogenetic reaction.

Within the last ten days, I have been in Pittsburgh, where they have a large number of infected, compound wounds involving bones and soft tissues, and I find that they are there using sulfathiazol to considerable advantage, placing in the wound, every day, a small amount of the powder at the time of dressing. They are unable, from my observation, to make any definite statement about its final value, and I believe that the question of drainage and the amount of freedom with which the secretion may run out of the wound has a great deal to do with its action.

DR. FRANK L. MELENEY (New York, N. Y.): I do not feel that I have any right to say anything about the clinical use of these drugs in crystalline form as my experience has been very limited. I have used them almost entirely by mouth or intravenously, but I should like to mention a few points

in Doctor Key's paper, and I hope that he will not mind if I find some faults with the experimental work.

First, his experimental set-up does not simulate clinical conditions. The subject of his paper being "contaminated wounds," I hardly think it is fair to limit the bacterial contamination in his experiments to the staphylococcus. I presume that he is considering principally traumatic lesions, such as accidental wounds, and we know that they are contaminated with a number of different organisms, the staphylococcus, of course, being one, almost invariably, but the hemolyticus streptococcus and the anaerobic bacteria are perhaps of more importance. Of course, in accidental wounds, the organisms are probably present in very much smaller numbers and may have a symbiotic effect which pure cultures will not have. In other words, he should have contaminated his experimental wounds with mixtures of organisms commonly found in accidental wounds.

I also think that he should have taken into consideration the trauma that exists with accidental wounds, and similarly traumatized his experimental wounds.

Of course, one of the prime requisites of proper treatment of accidental wounds is the removal of all injured tissue, foreign bodies, *etc.*, but in most wounds that are so treated there is still some damaged tissue remaining after débridement. We know that the sulfanilamide group of drugs, even under the best conditions in the test tube, do not have a bactericidal action and only have a bacteriostatic action if the organisms are present in very small numbers. Furthermore, they are particularly inactivated by injured tissue.

I think it is very difficult to produce experimentally, in animals, conditions similar to those which exist in human wounds clinically. Therefore, it is very difficult to draw conclusions from animal experiments of this kind and I do not think that Doctor Key's results justify the conclusions at which he has arrived. After all, perhaps the most important point I can make is that the final evaluation of these drugs will have to be made from clinical observation of a great many cases. I think it should be kept in mind, however, that if crystals are put into wounds they do act as foreign bodies and, to that extent, are not only irritating but mechanically separate the wound surfaces. Furthermore, there is always the possibility of toxic effects which should be kept in mind and carefully studied if any clinical experiments are to be recorded.

Finally, let me make a plea for adequate bacteriologic studies in these clinical investigations. There should be a complete bacteriologic analysis of the flora, both aerobic and anaerobic, of all wounds treated either locally or generally with these drugs. Then we will learn what these drugs will really do against every species and every combination.

Dr. Owen H. Wangensteen (Minneapolis, Minn.): I wish to speak briefly concerning the value of local implantation of sulfanilamide about the operative site in experimental gastro-intestinal surgery upon the dog. There has been a lively local interest relating to the implantation of sulfanilamide in wounds since Dr. N. K. Jensen, of the Minneapolis General Hospital, and his associates first pointed out the efficacy of employing sulfanilamide in this manner as a bactericidal and bacteriostatic agent.

During the current year, Dr. Richard L. Varco has made a very interesting application of the use of local implantation of sulfanilamide. Upon completion of gastro-intestinal anastomoses of varying kinds, Doctor Varco has been placing 4 to 5 Gm. of sulfanilamide about the suture. He has now an operative series of between 40 and 50 operations, many of which involve

triple anastomoses, such as the interpolation of a short segment of the terminal ileum and ascending colon between the pyloric outlet and the jejunum to imitate the function of the Dragstedt valve, with transfer of the biliary and pancreatic secretions to a lower level in the bowel (duodenal drainage according to the Mann-Williamson method), transection and end-to-end anastomosis of the esophagus, the establishment of Pavlov gastric pouches, gastric resections and similar procedures with only three deaths in the group (none with peritonitis).

This is really an unusual accomplishment for any surgeon. Markowitz, a pupil of Frank Mann, has said quite appropriately: "If an operator has mastered the technic of resection in dogs, he need not doubt his ability to make a safe anastomosis of the human intestine. When a surgeon can perform on dogs the operation of functional exclusion of the duodenum, known as 'duodenal drainage,' with a mortality of only 20 per cent, we should say that he has mastered the technic of intestinal anastomosis" (Experimental Surgery. William Wood and Company, 1937, p. 73).

The difficulties in the dog are well-known to all who have had an extensive experience in this type of surgery. Doctor Varco and I have been able to confirm Markowitz's observation, and from my own experience, I have the impression that similar anastomoses on man may be carried out with definitely lesser risk.

Even with employment of closed or so-called "aseptic" anastomoses in the dog, leaks may occur where the intestine is punctured with the needle, even though fine needles and silk are used. It is very discouraging to spend three to four hours making a complicated anastomosis in the dog to find, in 48 hours, that the anastomosis appears to leak in many places, particularly when everything seemed in order on completion of the procedure.

It is Doctor Varco's impression that the local implantation of sulfanilamide about the anastomosis exerts a bacteriostatic effect upon the bacteria which escaped through needle punctures of the intestinal wall. The presence of pathogenic bacteria on the peritoneal surfaces of the anastomosed segments, Doctor Varco feels, interferes with fibrin formation and stops the healing process. Local implantation of sulfanilamide holds the bacteria in check, preventing the lysis and destruction of fibrin, thus permitting the healing process to continue normally. Doctor Varco failed to observe a similar protective influence when the sulfanilamide was administered subcutaneously.

It is well known that the dog tolerates relatively larger doses of sulfanilamide than does man. Further, the dog does not acetylate a portion of sulfanilamide administered, as does man. Consequently, in the dog, all of it is available for bactericidal and bacteriostatic purposes. We have used sulfanilamide in this manner in colon resections, implanting usually 4 Gm. about the anastomosis and 2 Gm. in the abdominal wall above the peritoneum. The blood levels of sulfanilamide in man, following such implantation, come up to maximal levels in two to three hours' time. If the sulfanilamide could be implanted locally in oil, permitting even slower absorption, the protection afforded might be enhanced. It does appear that local implantation of sulfanilamide in the peritoneal cavity of the dog, about complicated gastrointestinal anastomoses, is a worthwhile procedure.

DR. J. DEWEY BISGARD (Omaha, Neb.): I would like to report some experimental work which we have carried out similar to that just reported. Our results, as far as soft tissue healing was concerned, were exactly as those reported, but we also carried out observations relative to the healing of fractures, and we found that the healing of fractures in the presence of large

quantities of sulfanilamide (these were all carried out with sulfanilamide) progressed at a perfectly normal rate and there was as prompt healing in those in which there was sulfanilamide present and as thorough healing as those of the controls.

Dr. Henry F. Graham (Brooklyn, N. Y.): Experimental work is valuable, but we should not forget the clinical side also. Many of us are clinical and not experimental surgeons. We should not forget the work that Doctor Garlock has done: He demonstrated, in his large colon surgery, that he could perform 25 operations in succession with a little skin infection in one only by the use of sulfanilamide by mouth or subcutaneously. We must not forget the article of Ravdin's, which came out in the January number of the Annals of Surgery, on the use of sulfanilamide subcutaneously in ruptured appendicitis cases and the remarkable results that he has obtained by its use subcutaneously.

We, personally, were interested in that, and within a few days after that article appeared a four-year-old child was brought into the hospital with a ruptured appendix and a large amount of fecal matter in the abdomen. The appendix was removed, a tube was inserted, and sulfanilamide was immediately started subcutaneously. No sulfanilamide was used locally in the wound or in the pelvis. Inadvertently, about the third day after the operation, this child reached under her dressings and pulled the tube out—and it was never reinserted. She went on to a complete recovery without any secondary operation.

We had a man with a ruptured appendix, and a fulminating peritonitis, and great abdominal distension. For days, he got sulfanilamide subcutaneously, and at autopsy—he died of pneumonia—there was no evidence of any peritonitis whatsoever. It had entirely cleared up.

We began by using sulfanilamide subcutaneously but found that the cost was prohibitive in the ampules, so, finally, in looking over a Winthrop Chemical Company pamphlet, we found that if the powdered sulfanilamide was thrown into water which had been brought to a boil and then taken off the stove or the heater it was not disintegrated, and we have been using it since that time in that way. We throw the 8 Gm. into the liter of boiling water. That makes a day's dose for an ordinary individual, we will say of average weight, 150 or 160 pounds. The first dose usually consists of 4 Gm. to bring the blood sulfanilamide to the proper level, and then it is continued every six hours thereafter hypodermically and we have found it very efficient. It gives a blood level of about 7 mg. per cent.

In several instances, where we have had tubes for drainage, we have taken the drainage from the tubes and have estimated the sulfanilamide concentration and have been able to get a concentration of 7 to 8 mg. per cent in the pus and discharge from the tubes. This local use may be valuable, but it remains doubtful, as yet, whether the introduction of a foreign body directly into the wound is better than its subcutaneous or oral administration.

Dr. J. Albert Key (St. Louis, Mo., closing): I agree that it is of value subcutaneously. I think it is of more value implanted locally, however, because the concentration is greater. I wish there were some way that we could produce a uniform local infection in animals. Until we can obtain a uniform local infection in animals I do not know how we are going to test out the efficacy of drugs on such an infection.

In regard to the trauma to tissues in compound fractures, before you put in sulfanilamide you perform just as careful a débridement as you would

if you were not going to put in sulfanilamide. The idea of the sulfanilamide is that it is probable that a very mediocre surgeon can obtain just as good results or better with it than can the best surgeon without it; at least, that is my opinion.

We heard last night about the work in Spain. Many of you may have read Trueta's report of that work. I believe that his method has been adopted by the British Medical Association. I had some correspondence with Dr. T. P. McMurray and he was quite enthusiastic about Trueta's work. There is nothing new about it. Doctor Gurd showed a similar method to the American Orthopedic Association when they met in Toronto several years ago. He débrided compound fractures very thoroughly, taking out all questionable tissue, and used a very thin layer of "Bipp" on his wounds after getting them as dry as he could and then put on a plaster encasement. In Spain they have used gauze. I believe that if they had used sulfanilamide most of those same wounds could have been closed. It would have saved a tremendous amount of after-care, it would have saved scars, and the results would have been much better.

In regard to the foreign body reaction, that is the reason we prefer sulfanilamide to the other two drugs. We take care that we do not put in lumps of the drug. You can crush it in your fingers, as it clumps up after being sterilized, and scatter it thinly over the surface. Then I usually rub it over with my fingers. I do not think it lasts long enough as a mass to create sufficient foreign body reaction to be objectionable.

In regard to bone healing, I have used it routinely in bone grafts for the past year or more and have not had any trouble.

In regard to toxic effects of the drug, in the literature, the fatalities have come late in the course of treatment in people who have received from 34 to 100 Gm. of the drug. There have been no fatal toxic effects from amounts comparable to those used in wounds.

Doctor Harbison and I have been working on the healing of the stomach and intestinal wall since Doctor Graham and Doctor Bricker showed that with therapeutic doses of sulfanilamide there was some slight delay in wound healing. I think that we will find that probably there will be a little delay, but we still do not believe that this delay is going to be sufficient to contraindicate the use of the drug.

DEGENERATION AND RECOVERY OF AUTONOMIC NEURONS FOLLOWING ALCOHOLIC BLOCK

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PARAVERTEBRAL INJECTION of alcohol to produce functional interruption of autonomic conduction pathways has become a recognized therapeutic procedure. It has been employed in many cases as a substitute for sympathetic ganglionectomy. Clinical reports indicate that it is useful not only in the treatment of diseases in which exaggerated vasomotor tonus exists at the periphery, but also in the relief of visceral pain. Actual investigation of the histologic and cytologic changes which take place in the autonomic nerves and ganglia following alcohol injections have not hitherto been carried out. The present investigation has been undertaken to determine what changes take place in the nerves and ganglia in question, whether nerves may be blocked permanently by means of alcohol injections, and under what conditions restoration of function may be expected.

Mandl[2] (1925) advocated blocking of sympathetic ganglia and fibers by injection of procaine hydrochloride in proximity to the sympathetic trunk ganglia in patients suffering from angina pectoris. Swetlow[8] (1926, 1930) reported successful use of alcohol injections after diagnostic use of procaine for more permanent relief from pain in such cases. Mixter and White[3] (1928), White and White[10] (1928), and White[9] (1930) also successfully treated patients with angina pectoris by this method. In summarizing the results of the first 35 cases treated in this way at the Massachusetts General Hospital, White[9] (1935) found that 67 per cent obtained almost complete relief, 25 per cent showed moderate to marked improvement, and 9 per cent showed no improvement. According to Smithwick[6] (1937), paravertebral injection of alcohol is as satisfactory in the treatment of angina pectoris as any operation of this magnitude, and has become the treatment of choice.

Stern[7] (1930) and Patterson and Stainsby[4] (1936) reported definite improvement in cases of thrombo-angiitis obliterans of the lower extremities following alcohol injections of the upper third or fourth lumbar segments. In patients with Raynaud's disease, Flothow[1] (1931) and Patterson and Stainsby (1936) observed immediate results as good as those following sympathectomy. Reichert[5] (1933) reported 25 cases of intermittent claudication without gangrene which were materially benefited by paravertebral injection of alcohol following the diagnostic use of novocain. In cases of polyarthritis which showed vasomotor disturbances, Flothow (1931) and

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Patterson and Stainsby (1936) reported relief from pain and improvement in joint function and in the symptoms related to faulty circulation.

Materials and Methods.—Cats have been used exclusively in this investigation. Nerve block was produced by injecting, with a syringe, 2 to 5 cc. of 95 per cent ethyl alcohol into the region of the ganglion or ramus to be blocked. Paravertebral and direct injections were employed. In the former method, the needles were inserted in the back, using the vertebral spine as guides. In the latter, the lumbar and cervical ganglia and rami were exposed by means of incisions through the skin and underlying tissues, and directly injected. This procedure was used almost exclusively in the later part of the work, since it was found that the percentage of successful paravertebral injections was low, whereas the desired results were obtained almost constantly by direct injection.

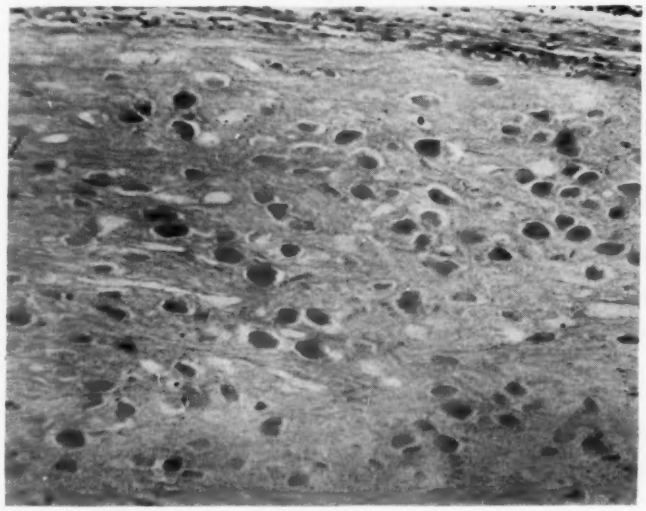

FIG. 1.—A section of a completely infiltrated sympathetic trunk ganglion removed after three days, showing the dead neurons and the absence of live interstitial cells in the area of infiltration. (×100)

The lumbar sympathetic ganglia and rami were used for the study of degeneration and regeneration of ganglion cells and postganglionic fibers following nerve block; the cervical sympathetic trunk was used for the study of the changes in the preganglionic fibers. To demonstrate the functional state of the blocked preganglionic fibers, faradic stimulation was applied to the cervical trunk between the site of injection and the stellate ganglion. The absence or presence of eye responses was correlated with the histologic findings.

The pyridine silver technic was used to demonstrate changes in ganglion cell bodies and their processes; the erythrosin-toluidine blue technic to demonstrate chromidial and nuclear patterns; and the osmic acid technic to demonstrate changes in myelin sheaths.

Histologic and Experimental Data.—Changes in Ganglia and Associated

Rami Infiltrated with Alcohol: Infiltrated ganglia, removed three days after injection and stained with erythrosin-toluidine blue, show marked degenerative changes (Fig. 1). The entire section reacts lightly to the acid stain and not at all to the basic stain. The only evidence of living cells is in the connective tissue surrounding the ganglia. The shrunken neurons show no cytologic structure except a very pale spherical nucleus. The widely dilated capillaries contain only fragmented blood cells making it evident that no blood flows through the infiltrated tissue. The histologic appearance closely resembles that of a recent infarct.

Occasionally, even though the ganglion is affected directly by the alcohol, the reaction is less intense, probably due to dilution of the alcohol by the

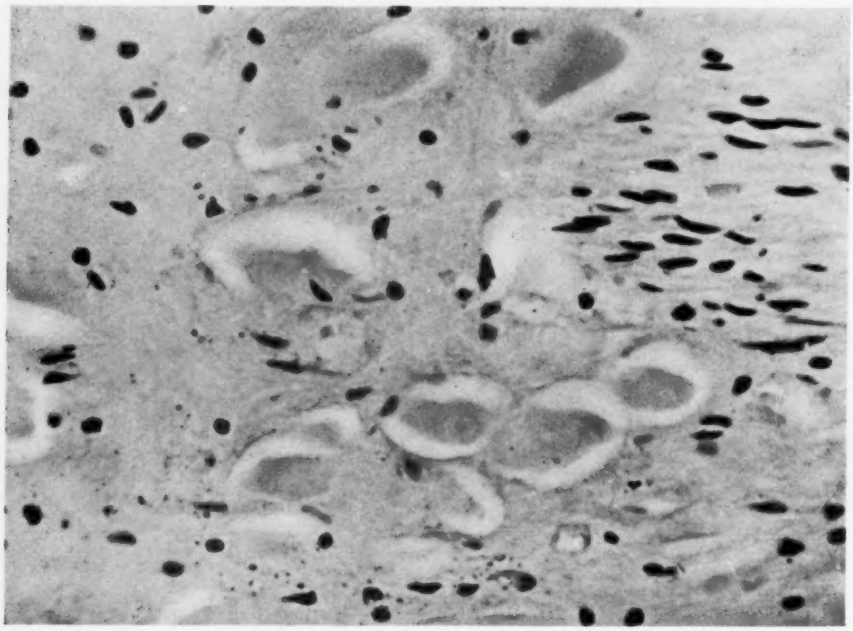

FIG. 2.—A section of an incompletely infiltrated ganglion removed after three days. The neurons have been killed but the more resistant interstitial cells have survived. (×400)

tissue fluids before it penetrated the ganglion, when the point of the needle was not sufficiently close to it. Under such conditions, the neurons seem to be the only cells affected. The remaining cells stain normally, the sheath cells being especially prominent (Fig. 2). Of all the cells in the ganglion, the neurons seem to be the most susceptible to the injurious effects of the alcohol, while the sheath cells are comparatively resistant.

Within the infiltrated rami of a blocked ganglion, the only tissue elements apparently intact are the connective tissue fibers of the endoneurium and perineurium. No cellular elements are visible except beyond the limits of the area of infiltration, where it is evident that the distal portions of the injured axons are undergoing typical wallerian degeneration.

Infiltrated ganglia retain the characteristics described above until after approximately 25 days, at which time cells, most of which are sheath cells, fibroblasts, and histiocytes, are found migrating into the necrotic area from the associated uninfiltrated rami. This is the only path by which cells can invade the tissue, since no functioning blood vessels are present. Wherever the invading cells are found in the ganglion, the neurons have either disappeared completely or are in a state of fragmentation. These necrotic neurons obviously are phagocytized by the invading cells. This phagocytosis is comparatively rapid as evidenced by the fact that very few neurons are present in the area occupied by the free cells (Fig. 3). Small blood vessels

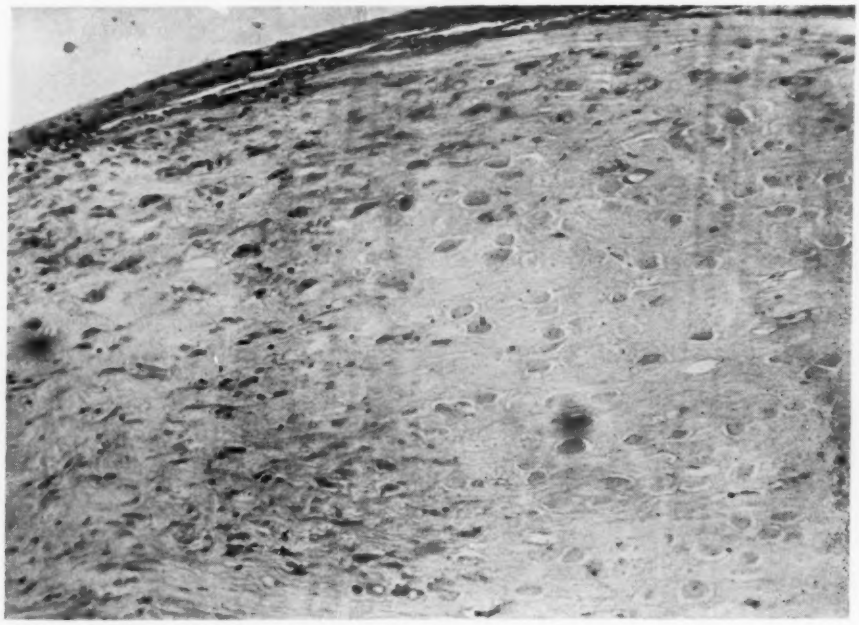

FIG. 3.—A section of a completely infiltrated ganglion removed after 30 days, showing phagocytosis of the dead neurons by the cells migrating into the area from an associated ramus. (×100)

grow into the tissue along with the migrating cells and reestablish circulation in the area.

In ganglia which are incompletely infiltrated, the ganglion cells undergo phagocytosis earlier than in those in which infiltration is complete. The cells of the interstitial tissue, being more resistant than the neurons, are not killed; consequently, phagocytosis of the ganglion cells begins at once, whereas, if infiltration is complete, phagocytic cells must first wander in from the uninfiltrated area.

In ganglia and rami removed 40 to 60 days after infiltration and stained with erythrosin-toluidine blue, the infiltrated region is distinguishable because of the light pale appearance of the tissue. Large phagocytes and young fibroblasts are present in abundance, but neurons are lacking (Fig. 4). At

approximately 40 days, silver preparations show a loose tangled skein of fibrils and collaterals of regenerating axons within the scar tissue at the site of injection. This is probably due to the absence of protoplasmic bands of sheath cells, and to the density of the connective tissue of the scar. By 60 days, however, sheath cells have become arranged in definitive protoplasmic bands in which regenerating fibers grow through the ganglion in more or less direct courses. It is evident, therefore, that some of the early regenerating fibers are lost in the scar tissue, due to the absence of previously established pathways, while later ones grow along the newly formed protoplasmic bands and reach the peripheral segments.

FIG. 4.—A section of the connective tissue mass which represents a completely infiltrated ganglion after 40 days. (×100)

Changes in Rami Alone Infiltrated with Alcohol.—Blocking preganglionic fibers by infiltrating the communicating rami does not directly affect their cells of origin, since the latter are located within the spinal cord. Postganglionic fibers may also be blocked in this manner without directly affecting the ganglion cell bodies, especially since the ganglia of the sympathetic trunk are more resistant to the effects of alcohol than the rami. The degeneration of the axons of both preganglionic and ganglionic neurons distal to the site of injection, as has been stated above, is typically wallerian. The accompanying retrograde reactions in the cell bodies are apparent as early as three days after injection and increase in severity. In preparations taken 25 to 50 days after infiltration, many of these cells are dead or dying. They show marked turgescence, absence of nuclei, and acidophilic cytoplasm with marked neuronophagia. Of the remaining neurons, some apparently

are normal, some show extreme chromatolysis, and some, other regenerative changes.

These changes are observed up to 90 days after infiltration, at which time most of the remaining cells either are normal or show characteristic evidence of recovery. The decrease in the number of ganglion cells present causes the remaining neurons to be more widely separated from one another than in a normal ganglion.

Changes in Preganglionic Fibers of the Cervical Sympathetic Trunk Infiltrated with Alcohol.—Infiltrated portions of the cervical sympathetic trunk removed within a few hours after injection of alcohol show beginning dissolution of the myelin. After ten days, myelin degeneration at the site of injection goes on more rapidly than in the remaining portions of the trunk. Only small globules of myelin remain. Distal to this area, the myelin seems to follow the usual process of degeneration which takes place when an axon is severed from the cell body.

Regeneration of the myelin is very apparent at 90 days, at which time it is present on the regenerating axis cylinders for variable distances. The process once initiated is so rapid that in sections of the trunk taken at the inferior border of the superior cervical ganglion 170 days after injection the myelin sheaths are completely regenerated.

Duration of Cervical Sympathetic Block as Determined by Faradic Stimulation.—Within a few minutes after injection of the alcohol to produce block of the cervical sympathetic trunk, the effects upon the eye can be noted. The pupil is constricted and the nictating membrane extended. This syndrome signifies blockage of the nerve impulses, but to further test the completeness of the block faradic stimulation was applied to the trunk between the area injected and the inferior cervical ganglion.

In the one cat which was allowed to live 170 days after block was produced, the eye syndrome was still apparent but in reduced degree. When faradic stimulation was applied to the lower portion of the cervical sympathetic trunk, the pupil slowly dilated and the nictating membrane receded. These responses were considerably slower than in normal cats, but they demonstrate that functional connections in the superior cervical ganglion have been at least partially reestablished in 170 days.

SUMMARY AND CONCLUSIONS

The histologic and cytologic changes in the autonomic ganglia and nerves of cats following exposure to alcohol either by paravertebral injections of the sympathetic trunks or by direct application have been described. The changes which occur after infiltration of a ganglion differ from those which occur after infiltration of the rami. When a ganglion is infiltrated, a permanent block to all effectors innervated by the postganglionic fibers taking origin from it is produced, since the alcohol kills the ganglion cells. Phagocytic cells migrate into the infiltrated area from the adjacent uninfiltrated rami and remove the necrotic ganglion tissue. After 35 days there remains only a

connective tissue scar. When the rami alone are infiltrated, a temporary block is produced. The alcohol affects the axons at the site of infiltration by partially dissolving the myelin, if the fibers are myelinated, and interrupting the continuity of the axons so that the distal segments undergo typical wallerian degeneration. Retrograde changes also occur in the related cell bodies which cause some of the neurons to undergo complete degeneration; others suffer injury in lesser degree and eventually recover.

The nerve tissue in the area of infiltration may be completely destroyed, yet the fibrous elements of the connective tissue maintain the continuity of the ramus. Block of this type is less permanent than that produced by

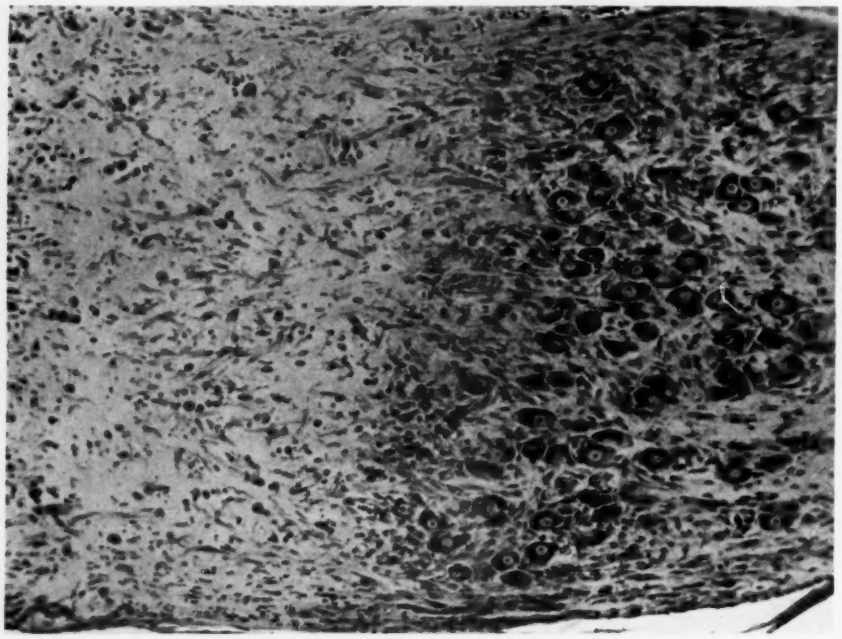

FIG. 5.—A section of a ganglion removed after 30 days, in which infiltration was only partial. Approximately one-half of the ganglion cells have been killed and phagocytized. (×100)

section of a ramus since, in the latter case, regenerating axis cylinders may span the gap between the proximal and distal segments of the ramus in order to reach the protoplasmic bands formed by the sheath cells of the distal segment. When a nerve is blocked by injection of alcohol, no gap is produced although a connective tissue scar is formed at the site of infiltration. Fibers penetrate this scar relatively early and with ease so that functional connections are established much sooner than following section of the nerve.

In order to produce permanent sympathetic nerve block by means of paravertebral alcohol injection, the ganglia must be infiltrated. If the needle is not inserted deeply enough, only the communicating rami may be affected, since the alcohol diffuses only slightly through the tissues. In the present investigation, infiltration of ganglia was affected only when the alcohol was

injected in their immediate vicinity. In some cases, only the rami were infiltrated even though the alcohol was injected close to the ganglia. In other cases, the infiltration was not sufficiently extensive to destroy all the ganglion cells (Fig. 5). These results indicate that the point of the needle must be practically adjacent to the ganglion or even penetrating it in order to insure complete infiltration. On the other hand, block of the rami is accomplished relatively easily. The variability of the results obtained by paravertebral injection in clinical cases may be explained at least in part on this basis.

Permanent block of sympathetic impulses to abdominal viscera by means of paravertebral injections of alcohol is impossible, since most of the neurons from which postganglionic fibers to these organs are derived are located in the prevertebral ganglia. The preganglionic fibers to these ganglia may be blocked by this technic but they are not prevented from reestablishing functional connections with the ganglion cells in question in a relatively short time. On the other hand, permanent and complete block of sympathetic impulses to the thoracic viscera or the extremities may be produced by paravertebral injection, since infiltration of the sympathetic trunk ganglia destroys the cells of origin of the postganglionic fibers involved.

The author is indebted to Dr. Albert Kuntz for helpful suggestions and criticisms throughout this investigation.

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BRIEF COMMUNICATIONS AND CASE REPORTS

ANGIOMA OF THE SKULL

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ANGIOMA OF THE SKULL is not a common condition as a careful review of the literature reveals only 26 cases. Of 1,831 neoplasms of the bone recorded in the bone registry, 13 have been hemangiomata. Of these only three involved the skull. In Christensen's[1] collection of 918 neoplasms of the bone, less than 1.5 per cent were hemangiomata. Geschickter and Copeland[2] report 12 cases of hemangioma of all types of bone in a group of 1,700 neoplasms of bone. These authors have called attention to the fact that most of the earlier reports failed to distinguish hemangioma from sarcoma and, in

FIG. 1.—Roentgenogram showing a bulging, soft tissue mass in the right frontal region.

1937, I[3] reported a case of angioma of the skull which was successfully removed, and there has been a total of nine tumors which were extirpated. Some authors have felt that roentgenotherapy or irradiation was the procedure of choice because of the extreme vascularity of the bone. There are two types, cavernous and capillary angiomata. The cavernous angioma is filled with large blood spaces resembling a cavernous angioma elsewhere in the body. The capillary type is one in which there are many small openings

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filled with blood and interspaced with a great deal of fibrous tissue. The first case of this series was of the capillary type.

Toynbee,[4] in 1845, was the first to describe angioma of the skull and, since then, there have been various references to this condition. The most recent has been the case reported by Anspach,[5] of a girl, age 11, who registered at the Children's Hospital in Chicago, in 1921, but the condition was deemed inoperable because of the extreme vascularity, and 15 years later she returned for irradiation. Irgens[6] reported an hemangioma of the skull involving the right petrous and occipital bones, which succumbed nine months after exploration and irradiation.

The basis of this report is the author's second case, which differs from the first in the fact that it was a cavernous hemangioma, and it was, also, successfully removed.

TABLE I

RÉSUMÉ OF ANGIOMATA OF THE SKULL AND ASSOCIATED LESIONS REPORTED BY VARIOUS AUTHORS

Author	Age	Sex	Location	Remarks
Toynbee[4]	19	M.	Parietal bone.	Died of tuberculosis.
Ehrmann[7]	40	F.	Left parietal bone.	Removal. Died of meningitis.
Cruveilhier[8]	38	F.	12 angiomata of skull; one of femur; one of third rib and one of both shoulders.	No details given.
von Rokitanski[9]			Angioma of parietal lobe.	None.
Morris[10]			Right parietal bone eroded.	Removal.
Stamm[11]	4 mos.	F.	Angioma; skin, muscles, bone, kidney, ovary, lungs, brain and skull.	Found at autopsy.
Pilcher[12]			Angioma; vault of skull.	Removed.
Ziegler[13]			Angioma of skull.	
Zajaczkowski[14]	38	F.	Frontal bone.	Removal. Tumor pulsated in center.
Schöne[15]	39	M.	Angioma: occipital bone.	Removal with recovery.
Major and Black[16]	34	M.	Angioma: each temporal bone.	Also angioma: liver and bilateral cystic adrenals found at autopsy.
Brandt[17]	47	M.	Angioma: left petrous bone.	Found at autopsy; also angioma: retina of right eye, cerebellum, cauda equina, kidneys, pancreas, spleen and questionably in the long bones. This was a report of von Hippel's case.
Kaufman[18]	76	M.	Angioma of skull.	Found at autopsy.
Cushing[19]	26	F.	Right parietal bone.	Successfully removed.
Lanari and Marque[20]			Two cases of angioma of skull.	Treated successfully with roentgenotherapy.
Fros[21]	70	F.	Angioma of frontal bone. In addition, multiple small angiomata of other cranial bones.	Found at autopsy.
Overend[22]		M.	Angioma of occipital bone.	Demonstrated roentgenologically and removed.
Geschickter and Copeland[3]			Five angiomata of skull.	One congenital angioma removed from skull and scalp of a 17 mo.-old female. Inner table of the skull intact.
Abbott[4]	3	F.	Angioma of right frontal area.	Successfully removed.
Anspach[5]	11	F.	Angioma of the left parietal region.	Not removed. Treatment with radium.
Irgens[6]	28	F.	Right occipital and petrous bone.	Inoperable. Treated with radium. Death nine months later.

CASE REPORTS

Case 1.—Referred by Dr. C. V. Edwards, of Council Bluffs, Iowa: D. T., female, age three, had had a normal birth and the rate of development was uneventful. However,

FIG. 2.—The angioma exposed at operation.

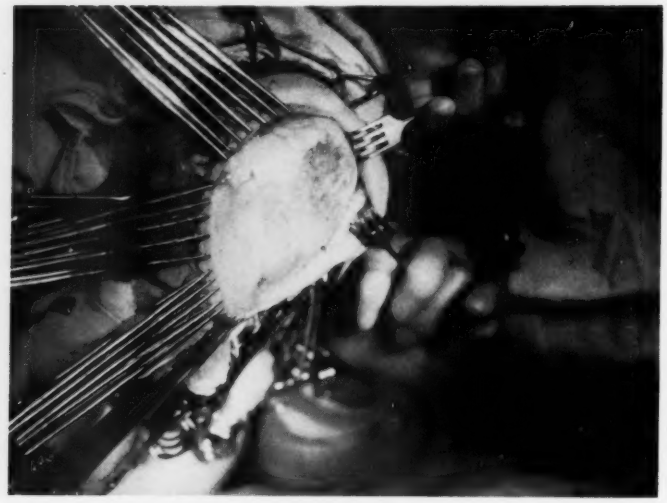

FIG. 3.—Illustrates the intact inner table of skull after removal of the angioma.

from birth it was noted that there was a defect in the right frontal region which would become larger when the child was reclining and smaller when it was in an upright position. The baby had a tendency to cry out sharply during the night. When two years

of age, roentgenograms were taken, and it was felt that the child was suffering from an encephalocele. Examination, June 23, 1936, revealed a well-developed and well-nourished child, 39½ inches tall, weighing 35 pounds. There was a bulging area in the right frontal region and palpation of this mass revealed the content to be filled with fluid. Auscultation failed to elicit any bruit. When the child would recline the mass would become larger and upon assuming the upright position it would become somewhat smaller. Other than the presence of large, cryptic tonsils, examination was essentially negative, as were laboratory tests of the blood and urine. Wassermann negative. Roentgenograms revealed a bulging mass in the right frontal region with a small line in the skull which might suggest an encephalocele. However, it was lateral to the midline.

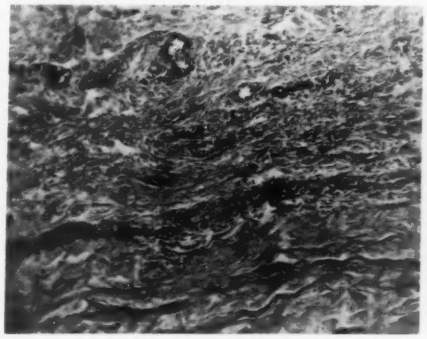

FIG. 4.—Photomicrograph of the tissue removed showing capillary angioma containing a marked amount of fibrous stroma. (×100)

The child was admitted to the Neurosurgical Service of Mercy Hospital and an attempt was made to reduce the size of the mass by spinal drainage. However, the spinal fluid pressure was 8 cm. of water, and withdrawal of 30 cc. of cerebrospinal fluid failed to affect the size of the mass. On June 26, 1936, under avertin anesthesia, a needle was introduced into the mass and bloody fluid was aspirated. A hockey-stick incision was then made around the mass, which was the size of a large frankfurter, measuring 10x4 cm. The skin was dissected away and a tumor mass was found to be attached to the skull. The mass was covered with periosteum.

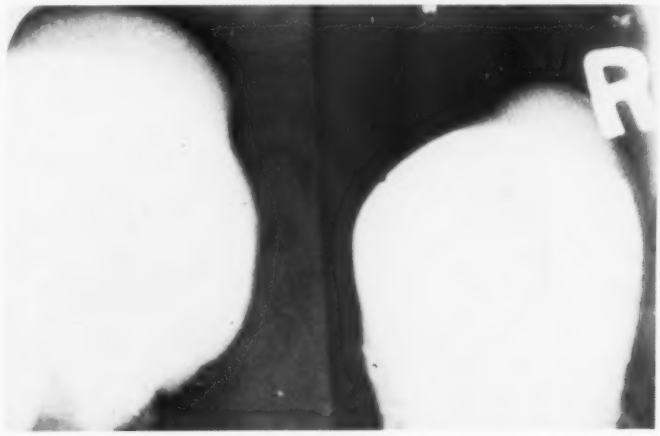

FIG. 5.—Roentgenograms showing a bulging, soft tissue mass in the right parietal region.

On carefully dissecting this mass free, many small veins were seen which communicated with the mass on the inner table of the skull which was found to be intact. The mass had completely replaced the outer layer of the skull. After its removal the bleeding of the bone was controlled with bone wax and an opening made in the inner table of the skull failed to reveal tumor tissue below.

Recovery was uneventful, and the child was dismissed from the hospital in ten days. Pathologic examination of the tumor showed it to be a capillary angioma of the skull.

A letter from the child's parents, February 26, 1937, stated that the little patient was developing normally and that there was no evidence of recurrence of the tumor.

FIG. 6.—The angioma exposed at operation.

Case 2.—Referred by Dr. Keith Chapler of Dexter, Iowa: W. G., male, age three weeks. The baby had been delivered in a normal manner but it was found that there was a large mass involving the entire right parietal region. The mass did not increase nor decrease in size during a period of three weeks' observation and, because of the same character of the tumor, it was felt that the child was not suffering from an hematoma of the skull. Examination revealed a well-developed and well-nourished baby of three weeks. Weight 8 pounds, 13 ounces. There was a large sausage-shaped mass, measuring 10x3 cm., involving the right parietal bone. Roentgenograms of the skull revealed a typical sunburst appearance of the inner table.

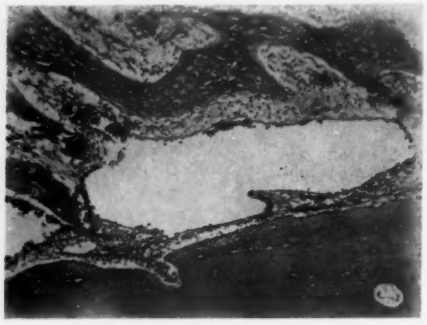

FIG. 7.—Photomicrograph showing a cavernous angioma. (×100)

Because of the first case, a preoperative diagnosis of angioma of the skull was made. On April 15, 1939, a hockey-stick incision was made around the bulging mass and the scalp was reflected over the tumor. There was a bluish tumor, covered with periosteum, which was removed from the skull.

The skull was irregular and there was a typical sunburst appearance of the parietal bone, measuring 3x5 cm., which was removed. Pathologic examination revealed the tumor to be a cavernous angioma.

The child made an uneventful recovery and, when seen September 22, 1939, was developing in a normal manner; there was no evidence of recurrence of the tumor.

CONCLUSIONS

Angioma of the skull is distinguished by the soft compressible tumor mass which sometimes is reduced in size when the patient assumes the erect position and enlarges, to a degree, when in the reclining posture. Roentgenograms reveal a so-called "sunburst" appearance, signified by radiating lines. This is more true in the adult than in children and it is my opinion that these may be removed surgically if extreme care is used in the control of hemorrhage during the operation.

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RESULTS OF SILK TECHNIC IN OPERATIONS FOR HERNIA*

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SILK SUTURE material in the surgical repair of hernia is not new. As long ago as 1890, Bassini used silk in his repairs but, in 1892, discarded it in favor of chromic catgut because of his infection rate and sinus formation. In 1899, W. B. Coley recorded his objection to the use of silk. He reported 400 operations upon children, with six recurrences—three with the Bassini method and three with other methods. There were three deaths; and he states primary union occurred in 373, slight suppuration in 15, and considerable in nine. There were two silk technic cases in his series; one of which resulted in marked suppuration, with extrusion of the sutures, and in the second case primary union occurred but a sinus developed later. Both cases recurred in four months. As a result, Doctor Coley stated he was opposed to nonabsorbable sutures.

Had it not been for the enthusiasm of Halsted and his followers, the use of silk might possibly have been completely discarded. In spite of its general lack of popularity at that time, Halsted continued to use silk; and in recent years there has been a general revival of its use.

Silk has a definite advantage over absorbable suture material in that the sutures remain in place until the wound has completely healed. The use of silk presupposes meticulous care in the handling of the tissues, the use of fine suture material, careful ligation of bleeding points without including great masses of tissue along with the vessels, and by using fine suture material the structures must be approximated without tension. All these factors, absolutely necessary in using silk, are, in themselves, of the utmost importance if primary union is to be obtained. Unless all these necessary precautions are carried out, wound infections and sinuses are likely to result.

In October, 1934, silk as a suture material was begun on the First Surgical Division at the Ruptured and Crippled Hospital, Dr. Carl G. Burdick, Director. At first, only the occasional case was operated upon with silk technic, but as time went on it was employed on more and more of the hernia cases until at the present time only rarely is any other material used.

From October, 1934, to January 1, 1938, 777 hernia operations have been performed on the First Surgical Division at the Ruptured and Crippled Hospital. All operations have been performed by a group of six visiting surgeons and 12 house surgeons.

* Read before the Clinical Meeting of the New York Surgical Society, March 22, 1939. Submitted for publication May 23, 1939.

Before proceeding to a follow-up on these cases, it might be interesting to mention the follow-up results on previously reported series of cases.

Roux reported 54 recurrences in 324 cases traced longer than two years, or a 15 per cent recurrence rate.

Bassini: Seven relapses in 251 cases, or 2.7 per cent.

Halsted: Twelve relapses in 221 cases, or 5.4 per cent.

Kocher: Fifteen relapses in 171 cases, or 8.7 per cent.

Coley: Five relapses in 514 cases (one to seven years), or 0.9 per cent.

Bloodgood, in 1900, reported 394 operations for inguinal hernia at the Johns Hopkins Hospital; 238 of these, personally examined by Doctor Bloodgood, showed six recurrences.

Hoguet, in 1918, reported 3,725 cases of simple inguinal hernia with 14 relapses, or 0.39 per cent.

In 1919, Masson, of the Mayo Clinic, reported 7,016 inguinal herniae repaired with 20-day chromic catgut with less than 1 per cent recurrence.

In 1935, from New York Hospital, Glenn and McBride reported 500 herniae repaired with silk technic with 2.3 per cent recurrence in indirect inguinal group, and 6.21 per cent in the direct inguinal group.

In February, 1939, Longacre, reporting from Presbyterian Hospital, found a 5.5 per cent recurrence rate in all indirect inguinal herniae, with 9.8 per cent recurrence rate in chromic gut cases, and 2.16 per cent in the silk technic cases. In the direct inguinal cases a total recurrence rate of 6.4 per cent with a 13.6 per cent rate in the chromic cases, and 4.49 per cent in the silk technic cases.

In most of the above reports the details of the follow-up are not mentioned. In our series, only those cases seen, and personally examined, at least one year after operation are included. It is our feeling that differences in follow-up methods account for the wide variations in the recurrence rate of the above mentioned series of cases, as the type of operation, except for slight modifications, was essentially the same in each series.

Table I summarizes the results of the 777 operations performed on the First Surgical Service of the Ruptured and Crippled Hospital from October 24, 1934, to January 1, 1938.

TABLE I

Cases Followed One Year or More: 552, or 71 Per Cent.

Primary indirect hernia: 334, with 22 recurrences, or 6.3%
Primary direct hernia: 118, with 15 recurrences, or 12.7%
Primary ventral hernia: 22, with 1 recurrence, or 4.5%
Recurrent inguinal hernia: 40, with 15 recurrences, or 37.5%
Recurrent ventral hernia: 7, with 1 recurrence, or 14.5%
Primary femoral hernia: 20, with 3 recurrences, or 15.0%
Recurrenf femoral hernia: 1, no recurrence.

It is interesting to note the infection rate on these cases. We have

classified as infections, every stitch abscess, hematoma, and sinus formation, as well as the deep abscesses—whether or not they prolonged the patient's stay in the hospital. During the first year, from October, 1934, to October, 1935, 85 operations were performed, with three infections, or 3.8 per cent. In the second year (October, 1935, to October, 1936), there were 328 operations, with 11 infections, or 3.3 per cent. In the third year, from October, 1936, to January 1, 1938, there were 364 operations, with five infections, or 1.3 per cent. From January 1, 1938, to January 1, 1939, 614 operations were performed throughout the whole hospital (both service and private cases), with four infections, or 0.65 per cent. This gradual reduction in the infection rate we ascribe, to a great extent, to increasing familiarity with the silk technic.

DISCUSSION: DR. FRANK L. MELENEY (New York) said that the use of silk had come into Presbyterian Hospital from Johns Hopkins in a roundabout way through Peking, China. Adrian Taylor, one of Doctor Halsted's assistant residents, took it over to Peking in 1920, and Doctor Meleney brought it back in 1925. It took four years for him to persuade the Presbyterian staff to use silk in clean cases. This was accomplished in 1930, by a clear demonstration of the very decided difference in the incidence of wound infection between cases sutured with silk and those sutured with catgut. This fact was first demonstrated in a series of thyroid cases, where there had previously been an incidence of infection around 15 to 20 per cent. This encouraged the staff to use silk in a group of herniae and, in 1931, the same unimpeachable evidence proved the superiority of silk over catgut. In 1932, the fracture service took it up, and since then the use of silk has spread to practically all of the clean cases. Since 1925, wound infections and hematomata have been rigidly kept track of, and with any group of cases, every year since 1930, the silk cases have had a lower incidence of both of these complications than the catgut cases. Longacre's recent statistics from Presbyterian Hospital now show that hernial recurrence is also less in silk cases. Of course, there have been individual cases in which silk ligatures have come out of persistent sinuses, but in spite of that the staff at Presbyterian Hospital has become converted to the silk technic because all of the statistics prove it the best, beyond the shadow of a doubt. Doctor Meleney said he had often wondered why Halsted never supported his preference for silk over catgut by comparable statistics. Doctor Heuer has explained this by stating that they had no catgut cases to compare. It is interesting and significant that now reliable statistics are coming from several different clinics which fully support Halsted's clinical observations.

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MEMOIR

HOMER GAGE

1861–1938

THE CAREER of Homer Gage was the natural and well-nigh inevitable result of the ancestral traits which he inherited from forebears whose intelligence, enterprise, frugality, public spirit and love of liberty contributed to the evolution of the Massachusetts Bay Colony into the New England of to-day.

HOMER GAGE, M.D.

His earliest colonial ancestor settled in Ipswich, Mass., in 1633, and was the progenitor of substantial citizens who fought in the Indian and Revolutionary Wars. The first physician in direct line was his grandfather, Dr. Leander Gage; his father, Dr. Thomas H. Gage, a noteworthy physician of his day, became president of the Massachusetts Medical Society.

Homer Gage was born in Worcester, Mass., October 18, 1861, and died July 3, 1938, at the age of 77, having spent his long life in fruitful activities of the most varied nature in the service of the community of his birth. From the Worcester High School he entered Harvard, graduating from the college with high honors in 1882, and from the Medical School in 1887, and then secured clinical training as surgical house officer at the Massachusetts General Hospital, and at the Children's and Lying-In Hospitals. Though his abilities must have tempted him to settle in the larger metropolis near academic shades, he chose to return to Worcester where he entered general practice in 1888 and speedily began to devote himself to surgery whose attractions were being rendered irresistible by the tardy universal acceptance and adoption of the principles of asepsis and antisepsis. He served the three principal hospitals of Worcester—the City, the Memorial and St. Vincent's—in almost every capacity: surgical, consulting and fiduciary. He knew the satisfactions and felt the obligations of organized medicine, serving as councilor of the Massachusetts Medical Society for 48 years, and as chairman of important committees. It is needless to enumerate the national and local organizations to which he belonged, in most of which his unusual abilities brought offices and honors.

Doctor Gage was elected to Fellowship in the American Surgical Association in 1910, and served as vice-president in 1919. He presented four papers before the association: In 1915, on "Acute Appendicitis as a Complication of Typhoid Fever"; in 1919, on "Postinfluenzal Abscess in the Sheath of the Rectus Muscle"; in 1921, on "End-results of 100 Cases of Cancer of the Breast"; and, in 1923, on "Embryoma of the Kidney." He valued his membership highly, as attested by his constant attendance during his active years. During the World War he rendered distinguished service as chief of the Surgical Division at the Base Hospital at Fort Devens from November, 1917 to February, 1919, retiring with the rank of lieutenant colonel.

On the medical stage of Worcester, Doctor Gage was, for years, a dominant and beneficent figure. He was a skillful and conservatively bold surgeon and a widely sought counselor. He felt great personal interest in the constantly increasing group of young men who, as house officers, became his disciples in the three hospitals which he served, and many of whom became his junior colleagues in practice. These men recorded their obligation by saying: "We feel that we have been brought up by him in our professional life. His personal influence, guidance and consultations, his able working out of surgical problems, wise conservatism and technical skill will remain as a lasting inspiration." Some 30 papers in medical periodicals testify to his sense of obligation to contribute to the education of the profession at large by communicating what he had learned in clinical practice.

During his mature and later years his versatile qualities and wide interests caused him to be drafted into the service of education, philanthropy and finance. He was a perfect example of that quite rare individual, an able physician with equal competence in the world of business and the social

sciences. He became the trusted president, director or trustee of important financial and philanthropic undertakings almost too numerous to mention. He was an overseer of Harvard University for 12 years. He was president of the Community Chest of Worcester for 17 years and directed the raising of its funds. He was a devoted friend of France and was largely instrumental in raising the large sum needed to build and endow the American Dormitory of the University of Paris—a service which won for him a decoration as Commander of the Legion of Honor. A resolution drawn up by associates said: "His service to his fellowmen was of a caliber that few men equal and none surpass; it is hard to imagine a busier and more useful life; . . . philanthropy was his ruling passion."

A man can scarcely lead such a life without the sympathy of a loyal wife which was the privilege of Doctor Gage. His later years were saddened but not crushed by the loss of an only child, a son, who survived the perils of the World War only to die prematurely without issue.

DAVID CHEEVER, M.D.

SECTIONAL MEETINGS OF THE AMERICAN COLLEGE OF SURGEONS

Dates	City	Headquarters Hotel	Participating States
March 10 11 12	Minneapolis Minnesota	Nicollet	Minnesota, North and South Dakota, Iowa, Nebraska, Montana, Kansas, Wisconsin—Manitoba
March 17 18 19	Pittsburgh Pennsylvania	Wm. Penn	Pennsylvania, Ohio, Virginia, West Virginia, Delaware, Maryland, New Jersey, New York, District of Columbia
March 26 27 28	Salt Lake City	Utah	Oregon, Washington, California, Nevada, Idaho, Wyoming, New Mexico, Arizona, Colorado, Montana, Utah

Hospital conferences will be held in connection with each of these meetings. Fellows of the College, members of the medical profession at large, and persons interested in the institutional care of the sick and injured, are invited to the Sectional Meetings; on the final evening of each meeting, a Meeting on Health Conservation to which the public is invited, will be held.

BOOK REVIEWS

ORTHOPEDIC OPERATIONS. By ARTHUR STEINDLER, M.D., Professor of Orthopedic Surgery, University of Iowa. Springfield, Ill.: Charles C. Thomas Co., 1940.

THIS WORK is based upon the author's personal experience, which seems to the reviewer to be a unique asset rather than a liability, as the author fears. Few will take exception to his claim that personal conviction, backed both by reflection and by experience, is the mainspring of unbiased teaching.

The object has been to combine into a triad the indications, technic, and his end-results in the several phases of operative orthopedic treatment. This statement of a modest man does not, however, indicate the scope of the work, for it is far more than a manual of orthopedic technic. Thus, under indications, in Chapter I, he discusses the biology of functional restoration under two headings: (a) Form and alignment; and (b) function—but he again divides the subject of form and alignment into two subtitles: (a) Functional adaptation of bone; and (b) functional adaptation of the soft tissues—the nature of contracture. Under function, he considers four subjects: (1) The relation between stability and mobility; (2) active stability; (3) passive stability, which is subdivided into (a) bone and (b) soft tissues, and (c) joints; and (4) the principles of operative reconstruction of muscle balance.

Chapter II is devoted to special surgical risks in orthopedic operations, and it is divided into three parts with 13 subdivisions.

There are 26 chapters in all, with text prepared with meticulous care, and to this is added an author's index and 22 pages of a subject index.

The publishers have fully attained, in this volume, their desire "to present books that are satisfactory as to their physical properties and artistic possibilities." The binding, paper, type and the illustrations are worthy of the unusual text.

The originality of the arrangement of the subject matter, however, was confusing to

the reviewer, even though he followed the warning of the author that if the book is to be used for reference the reader should first consult Part III for the discussion of the diagnosis, then Part II, for a description of the technic of the contemplated operative procedure, and, finally, Part I, in order to obtain an estimate of the surgical hazards involved and to try to anticipate them, or to treat them if they cannot be prevented. This may be the logical way to prepare and to use a book, but it certainly is not the usual way.

WALTER ESTELL LEE, M.D.

CANCER—A MANUAL FOR PRACTITIONERS. Boston: Committee of the Massachusetts Medical Society, 1940.

THIS BOOK was prepared for the practitioners of medicine in Massachusetts by a special committee of the State Medical Society, and financed jointly by the United States Public Health Service and the Massachusetts Branch of the American Society for the Control of Cancer.

It is indeed a fitting legacy from the men to whom it is dedicated:

Robert B. Greenough, M.D., surgeon, whose work to further our knowledge of malignant disease, and to control its ravages through education and modern methods of treatment, places him among the leaders of his generation in this field.

George H. Bigelow, M.D., who, as former Massachusetts Commissioner of Public Health, instituted the plan for cancer control, now in successful operation in that State, and which, in principle, is being generally adopted in other communities.

In the 44 chapters the reader will find a presentation of this problem which we feel would have met with the approval of these two surgeons.

The early chapters, which deal with such general subjects as the historic trend, epidemiologic aspects and principles of treatment, serve as a prologue to the 27 chapters dealing with the special tissues and organs. The closing chapters are concerned with such subjects as leukemia, Hodgkin's disease, industrial cancer, tumors of the endocrine glands, and the care of the patient with advanced cancer.

It will thus be seen that it is all inclusive and should be of incalculable value, not only to the rank and file of the medical profession for whom it was prepared, but also to the specialist as a reference book.

The discussion of the historic trends, by Eleanor MacDonald, makes fascinating reading and presents evidence that the problem of cancer has staggered and intrigued man from the beginning to the present time. She refers to an ancient Indian epic, the Ramayana, of about 2000 B.C., in which a disease was described that was evidently cancer. At that early date, the Indians were the first group to leave records of a systematic presentation of disease. Though their knowledge of anatomy was quite superficial, their surgery was daring, and radical extirpation of malignancy was practiced. It was the Egyptians who introduced arsenic in the form of a paste for the removal of malignancy, and the term cancer was first used by Hippocrates, but with the meaning of "benign tumor," while he employed the word "carcinoma" to describe malignant tissue.

Murphy, in his description of the trend in cancer research, concludes his article with the statement that the fundamental problem in the search for the genesis of cancer is the understanding of the factors responsible for the growth property of the cancer cell. In order to reduce this question to an experimental level, he assumes, with some evidence, that there is in the cell an interacting, balancing mechanism regulating growth and differentiation, and that malignancy represents a break in this mechanism.

Daland, in the chapter on "Cancer of the Breast," offers a statistical report from the Pondville Hospital, which deserves widespread publicity to the lay public as well as to the medical profession because of their encouraging results.

Sixty-nine per cent of their patients have shown five-year cures when the axillary nodes have not been involved. When this same group was followed from five to 11 years,

the percentage dropped only to 64 per cent. Thirty-two per cent of the cases were apparently well after five years in the group in which the malignancy extended beyond the breast at the time of the operation; there was a fall to 30 per cent only when these patients were followed from the fifth to the eleventh years. The combined percentage of recovery in these two groups, of 45 per cent after five to 11 years, is indeed surprising.

Pemberton does not offer such encouraging results in his chapter on cancer of the ovaries, and although there is an expectant salvage of 35 per cent after five years post-operatively, only 11 per cent of these patients are free of the disease. The results are discouraging and earlier diagnosis must be made if we are to obtain any improvement.

Space will not permit a more detailed discussion of this compilation of our present knowledge of the subject of cancer, but we hope that this review will convince the medical profession that the report contains reference material from an authoritative source which should be in the library of every physician and surgeon.

WALTER ESTELL LEE, M.D.

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